UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF TEXAS
HOUSTON DIVISION

STEPHEN McCOLLUM, and SANDRA McCOLLUM, individually, and STEPHANIE KINGREY, individually and as independent administrator of the Estate of LARRY GENE McCOLLUM,	§	
PLAINTIFFS	§	
	§	
v.	§	CIVIL ACTION NO.
	§	4:14-cv-3253
BRAD LIVINGSTON, JEFF PRINGLE, RICHARD CLARK, KAREN TATE, SANDREA SANDERS, ROBERT EASON, the UNIVERSITY OF TEXAS MEDICAL BRANCH and the TEXAS DEPARTMENT OF CRIMINAL JUSTICE.	§	JURY DEMAND
DEFENDANTS	§	

PLAINTIFFS' CONSOLIDATED RESPONSE TO DEFENDANTS' MOTIONS TO STRIKE SUMMARY JUDGMENT EVIDENCE

Exhibit D

IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF TEXAS
DALLAS DIVISION

STEPHEN McCOLLUM, et al., Plaintiffs,	)	
v.	)	CIVIL ACTION
BRAD LIVINGSTON, et al., Defendants.	)	NO. 3:12-CV-02037

ORAL DEPOSITION

SUSI VASSALLO, M.D.

March 5, 2014

ORAL DEPOSITION OF SUSI VASSALLO, M.D., produced as a witness at the instance of the Defendant UTMB and duly sworn, was taken in the above-styled and numbered cause on the 5th day of March, 2014, from 10:03 a.m. to 5:06 p.m., before Dalia F. Inman, Certified Shorthand Reporter in and for the State of Texas, reported by computerized stenotype machine at the offices of The Edwards Law Firm, 1101 E. 11th Street, Austin, Texas 78702, pursuant to the Federal Rules of Civil Procedure and the provisions stated on the record or attached hereto.

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ALSO PRESENT:

MS. JENNIFER OSTEEN, UTMB LEGAL AFFAIRS

MR. LARS HAGEN, ATTORNEY, U.T. SYSTEM

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thermoregulation that best represents the body of literature for the last 25 years or so. However, there are, of course, other things that one reads that one doesn't write down in one's...

Q I don't want to interrupt you.

A Yeah. In one's bibliography.

Q Okay. So --

A Excuse me. Because I don't -- I want to be complete. There is also the Angola report where they asked for bibliography. I think that had another 80 or so. There's overlap in those. But I direct you to those as well. And those are also, of course, all in the literature, peer-reviewed, and publicly available.

So I brought a few of the articles here today. And a few of them I would have -- be able to make available to you to make the job easier through searching my computer. And then some of them are hard copies. They're all publicly available through the public library.

Q Okay. So to make sure I understand what you're saying. On the Exhibit 1, page 4 and 5 to your subpoena duces tecum, I actually went through and made little stars by the articles or the lecture materials that I was interested in. Some of them, I will tell you, I did try to get off of the Internet and was not able to

Q Okay. What else do you have? Let's just do it this way. Why don't you just show me everything that you have.

A Okay. This is the Angola report with bibliography at the back, and so those would also be important articles that I relied on in my opinion.

(Exhibit 3 marked)

Q (By Ms. Coogan) And the Angola report has been marked as Exhibit 3?

A Yes, it has.

Q Okay. I have to do it so that -- on the record.

What else you got?

A I've pointed out, these are -- some of these are some of those that I just happen to have. So --

Q Okay. Let me stop you right there and get it marked so we don't say "this" and "that."

(Exhibit 4 marked)

Q (By Ms. Coogan) So we just marked as Exhibit 4 a notebook that says "Articles On Heat." And what is this?

A So these articles are contained within the materials I've told you already --

Q Okay.

A -- which is these from expert. All of these

represent some hard copies of some of the key articles. And they're not even key, just some subset of these articles that I've referred to.

Q Okay. And is that a copy of your report?

A This is my CV.

Q Your CV.

(Exhibit 5 marked)

Q (By Ms. Coogan) Exhibit 5 is your CV?

A Yes.

Q Okay. Okay.

A Expert opinion.

(Exhibit 6 marked)

Q (By Ms. Coogan) Exhibit 6 is your actual report in this case?

A Yes.

(Exhibit 7 marked)

Q (By Ms. Coogan) Exhibit 7, what is this folder?

A This folder is not contained within the bibliography of the thermoregulation chapter and the Angola bibliography. These are a couple of papers on diabetes, their reviews, obesity, and hypertension.

Q Okay. And did you rely on these articles in forming your opinion in this case?

A Yes.

Q And I see you have plaintiff's second amended

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)
BRAD LIVINGSTON, et al.,)
Defendants.)

REPORTER'S CERTIFICATION
ORAL DEPOSITION OF
SUSI VASSALLO, M.D.
March 5, 2014

I, Dalia F. Inman, Certified Shorthand Reporter in and for the State of Texas, hereby certify to the following:

That the witness, SUSI VASSALLO, M.D., was duly sworn by the officer and that the transcript of the oral deposition is a true record of the testimony given by the witness;

I further certify that pursuant to FRCP Rule 30(f)(1)the signature of the deponent:

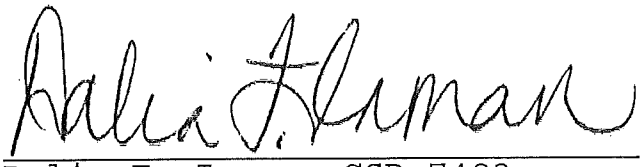
__X__ was requested by the deponent or a party before the completion of the deposition and returned within 30 days from date of receipt of the transcript. If returned, the attached Changes and Signature page

contains any changes and the reasons therefor;

_____ was not requested by the deponent or a party before the completion of the deposition.

I further certify that I am neither attorney nor counsel for, related to, nor employed by any of the parties to the action in which this testimony was taken. Further, I am not a relative or employee of any attorney of record in this cause, nor am I financially or otherwise interested in the outcome of the action.

Subscribed and sworn to on this the 13th day of March, 2014.



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UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF TEXAS
HOUSTON DIVISION

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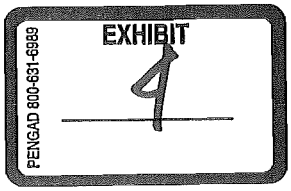
PLAINTIFFS' CONSOLIDATED RESPONSE TO DEFENDANTS' MOTIONS TO STRIKE SUMMARY JUDGMENT EVIDENCE

Exhibit E

Articles

ON Heat



Materials Relied Upon or Consulted

by

Dr. Susi Vassallo

HEALTH EDUCATION RESEARCH Vol.26 no.1 2011
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Correlates of hot day air-conditioning use among middle-aged and older adults with chronic heart and lung diseases: the role of health beliefs and cues to action

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Abstract

Extreme ambient heat is a serious public health threat, especially for the elderly and persons with pre-existing health conditions. Although much of the excess mortality and morbidity associated with extreme heat is preventable, the adoption of effective preventive strategies is limited. The study reported here tested the predictive power of selected components of the Health Belief Model for air-conditioning (AC) use among 238 non-institutionalized middle-aged and older adults with chronic heart failure and/or chronic obstructive pulmonary disease living in Montréal, Canada. Respondents were recruited through clinics (response rate 71%) and interviews were conducted in their homes or by telephone. Results showed that 73% of participants reported having a home air conditioner. The average number of hours spent per 24-hour period in air-conditioned spaces during heat waves was 14.5 hours (SD = 9.4). Exploratory structural equation modeling showed that specific beliefs about the benefits of and drawbacks to AC as well as internal cues to action were predictive of its level of use, whereas the perceived severity of the effects of heat on health was not. The findings are discussed in light of the need to adequately support effective response to extreme heat in this vulnerable population.

Introduction

Unusually hot weather is now recognized as a serious public health threat [1–3]. Recent heat waves have resulted in significant excess immediate mortality and morbidity [3–7]. Prolonged exposure to extreme heat, especially consecutive nights with high minimum temperatures, as during the 2003 heat wave in France and the 1995 Chicago episode, can provoke thousands of deaths [2, 5, 8]. Outside of these clearly demarcated 'heat waves', isolated days with temperatures only several degrees above average have been associated with higher-than-expected numbers of deaths from causes only indirectly related to overheating [9, 10]. Temperature change need not be extreme for heat-related risks to accrue: in the UK, older persons are at heightened mortality risk with any temperature rise above 17°C, and risk increases linearly or more as temperatures rise further [11]. Exposure to heat can exacerbate existing cardiovascular and respiratory conditions, resulting in increased hospital admissions and mortality [12, 13]. Indeed, the health effects of extreme heat persist long after temperatures have cooled: follow-up of heatstroke patients shows important declines in functional status, associated with prolonged hospitalization and earlier death [7]. It is expected that the public health problems attributable to extreme heat will be especially

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significant when combined with the increased vulnerabilities of an aging population and phenomena such as global climate change, urban heat islands and air pollution [1–5].

Older adults are at especially high levels of risk for heat-related illnesses, as are those living alone, without access to air-conditioning (AC), in urban areas, and with pre-existing health conditions [1, 3, 11, 14]. Mortality in the 2003 European heat wave was markedly higher among older adults living outside of institutions than in either the institutionalized elderly or in younger adults [15, 16] and was especially high among those with limited autonomy [14–17]. Nor do these risks apply only to the oldest old: heat-related mortality risk rises with increasing age after about 50 years [2].

Heat-related health risk is distributed inequitably across population groups. A recent review concluded that poverty is an important determinant of heat-associated mortality risk in American but not in European cities [2]. However, in the French heat wave of 2003, income was an independent predictor of mortality [17]. Coupled with the established link between ill health and poverty among older persons [18], older adults living in poverty may be the most vulnerable of all, for example, having to choose between purchasing food or buying and running an air conditioner [19].

Much of the excess mortality and morbidity associated with heat illness is believed to be preventable through the implementation of heat emergency plans and warning systems that lead to adoption of behavioral adaptations such as AC use and increased fluid intake [1, 4, 19]. In the Chicago heat wave of 1995, mortality was significantly lower among people who had working air conditioners in their homes or in their apartment lobbies or who visited air-conditioned places [8]. Although one study in the United States showed that over the period 1987–2000 cardiovascular mortality risks associated with cold temperatures have been constant among older persons [20], heat-related deaths in this population have declined. This was attributed to increased use of AC, in combination with other factors such as improved health care. In the French heat wave of 2003, older persons who visited air-conditioned or cooled places or who used cooling techniques and devices including air conditioners were less likely to die from heat-related causes than those who did not [14]. Exposure to AC, even for a limited time, thus can act as an important protective measure against heat-related mortality. Further, if AC or other recourse to personal cooling is employed, the need for activity reduction and supplementary hydration, other commonly recommended hot day protection behaviors, is much reduced [21].

Despite the effectiveness of such preventive measures, public recognition of the health risks associated with hot weather is low, as is adoption of effective preventive strategies. A study in four North American cities found that despite widespread awareness of heat advisories, only about half of urban residents adopted preventive actions [22]. Similarly, Kalkstein and Sheridan [23] showed that only 50% of residents who had heard heat advisories did something different on the days they heard them. Although evidence on the responses of the older adults to environmental health issues is more limited than that for the general population, some evidence suggests that older people are less likely to change behaviors in light of climate change [24]. Faced with environmental health threats, older people are equally unlikely or more unlikely than the general population to undertake protective actions, due to in part to perceptions of invulnerability [25]. And, one study has shown that likelihood of behavior change in response to heat advisories is related to age: whereas 67% of those aged 42–53 years reported making such changes, only 40% of those over 65 years reported doing so [23].

Given the potential to decrease their health risk from elevated heat events through more effective preventive action, it is of interest to identify the perceptions and beliefs that predict the adoption of such preventive behaviors among people at risk for heat-related illness.

The Health Belief Model

The Health Belief Model (HBM), originally proposed over 35 years ago [26–28], is one of the most widely used conceptual frameworks for the study of

health behavior. Over hundreds of studies, support has been found for the predictive role of all of its main components in the adoption and maintenance of health promoting and prevention behaviors. The HBM's components are (i) perceived susceptibility: an individual's perception of his or her risk of developing the health problem or being affected by the health threat; (ii) perceived severity or seriousness of the health threat. The combination of susceptibility and severity has been termed as perceived threat; (iii) perceived benefits of the preventive action, i.e. the perceived efficacy of the behavior in reducing the health threat's severity or the individual's susceptibility to it, as well as non-health-related benefits; (iv) perceived costs, barriers or disadvantages to the prevention action and (v) cues to action: the presence of information, reminders or bodily events that incite or encourage preventive action. A sixth component, self-efficacy, has been included more recently [28]. The model posits that each of these factors makes an independent contribution to adoption or maintenance of specific health behaviors.

Health beliefs and health threat response among older adults

Several studies have established the predictive validity of the HBM in older populations, for a variety of health issues. For example, older adults' influenza vaccination behaviors were predicted by perceived barriers, perceived benefits and perceived severity [29]. HBM components predicted sun-exposure protective behaviors in seniors [30], as well as health maintenance activities aiming to prevent health deterioration [31]. In a qualitative study of older women, low levels of perceived vulnerability were associated with a lack of attention to future health [32]. It is important to note that preventive health behavior in older adults seems more influenced by subjective than actual health status [33].

With respect to adaptation to climate change, no study has tested the Heath Belief Model in its entirety nor examined its utility in understanding preventive behaviors of vulnerable populations. However, studies have shown that a variety of beliefs—some corresponding or being very close to those of the HBM—predict adaptive behaviors. Studies of population response to natural hazard warnings have consistently shown that response is partly determined by perceived urgency of the threat [34]. In summarizing major covariates of hazard warning responses, Mileti and Sorenson [35] note that physical and social cues, perceived risk, perceived efficacy and proximity to the hazard all increase adequacy of response. Although there are few data on older people's beliefs in relation to heat protective behavior, those that exist suggest that HBM components may be useful predictors. A recent study of older people's (mean age of 80 years) perceptions of heat-related health risk in United Kingdom found that respondents did not necessarily perceive themselves as either 'old' or at risk, although they would have been identified as vulnerable by existing public health criteria. A minority was aware that their existing medical condition or medication increased their risk [36]. However, most reported adopting preventive behaviors, based on 'common sense' (p. 124), during previous heat waves. A survey of Phoenix, Arizona, residents found that while 93% of those over 65 years were aware of the heat warning system, older respondents were less likely than the youngest respondents to believe that heat was very dangerous to them—i.e. had low levels of perceived risk [23].

The study reported here tested the predictive power of selected components of the HBM for AC use among non-institutionalized older adults with two serious chronic health conditions: chronic heart failure (CHF) and/or chronic obstructive pulmonary disease (COPD). These individuals are highly vulnerable to heat threats: much of the excess mortality associated with extreme heat occurs through respiratory and/or cardiovascular failure [9]. Given the frequency of these two chronic conditions among older persons, and their association with social and economic disadvantage [18], individuals treated for CHF and COPD could be considered as representing members of the population vulnerable to serious consequences of heat exposure through both age and illness. The study addressed two main questions: (i) what proportion of high-risk middle-aged and older adults with

chronic heart and/or lung disease living at home display the preventive health behavior of air conditioner use? and (ii) what predictive role do health beliefs and perceptions play in that behavior? Although relying on self-reported behaviors, the study assessed usual behavior during high heat periods rather than behavioral intentions, which have been shown to be poor predictors of response to environmental health threats [34].

Methods

Sample and data collection procedures

We conducted a cross-sectional interview survey of middle-aged and older adults with CHF and/or COPD in Montréal, Canada. Patients were recruited from two CHF and three COPD clinics attached to two university teaching hospitals. Their participation was solicited either in the clinic waiting rooms (all patients were approached) or by telephone if patients were receiving treatment at home (patients meeting eligibility criteria were selected from clinic lists). Eligibility criteria were residence in the Montréal metropolitan area (population 3M), speaking either English or French and having a home telephone. Interviews were conducted either at home or at the clinic, according to respondents' preferences. Of the 343 patients solicited, 242 (71%) completed the interview. Four interviews with incomplete data were removed from the sample before analysis for a final n = 238. The interviews took place between 30 May 2005 and 6 October 2005. Daily mean temperatures during this period were 2.4°C above the long-term daily average [37].

Measures

The study questionnaire was developed in English following a literature review and consultation with professionals from two of the participating clinics. It was translated into French by members of the study team and back translated by a professional translator. The few differences were reconciled by the study team. The questionnaire was piloted in both languages. It consisted of 169 closed-ended items measuring among others, the HBM constructs. Perceived severity of health effects of heat waves was measured by nine items, for example: 'During a heat wave, if I do not protect myself from the heat, respiratory difficulties caused by the heat can lead me to be hospitalized' ($\alpha = 0.93$). Perceived susceptibility to heat effects was measured by three items, for example: 'Because of my state of health, if I do not protect myself from the heat, I am more likely to suffer from respiratory difficulties during a heat wave' ($\alpha = 0.88$). Perceived benefits of protective (AC) behavior was measured by five items, for example: 'During a heat wave, using an air conditioner at home allows me/would allow me to continue my daily activities as usual' ($\alpha = 0.86$). Perceived barriers to the health action were measured by eight items, for example: 'Does/could the following reason prevent you or limit you from using your air conditioner? It is not good for my health' ($\alpha = 0.84$). Perceived severity, susceptibility, benefits and barriers were measured on 4-point agree–disagree scales. Cues to action were measured by three items: 'Has your doctor or nurse ever told you that your health problem can make you more sensitive to the heat' (yes or no); 'Have you ever heard an extreme heat warning?', scored as 'no', 'yes, this summer' and 'yes, last summer or earlier'; and 'How sensitive are you to heat?', scored as very/somewhat/not really/not at all sensitive. Self-reported presence of an air conditioner (yes or no) and number of daytime and nighttime hours of air conditioner use (combined into a 24-hour measure then divided by 24) were used to assess the targeted behavior. Table I lists all items assessing health beliefs and cues to action. The SF-36 physical and mental health subscales [38] were also administered, as were items recording participants' sociodemographic and lodging characteristics. Not reported in this article but also assessed were knowledge of heat impacts, self-efficacy for adopting heat-protective behavior and attitudes toward heat advisories [39].

Analyses

An exploratory structural equation modeling (ESEM) approach was used. Recently developed

Table I. *Number and proportions of respondents falling into different response categories for each item of the scales assessing selected HBM components*

Items	Strongly disagree, *n* (%)	Disagree, *n* (%)	Agree, *n* (%)	Strongly agree, *n* (%)
Perceived benefits of AC				
'During a heat wave, using an air conditioner at home allows me /would allow me to':				
Q12: Continue my daily activities as usual (N = 238)	9 (3.8)	28 (11.8)	110 (46.2)	91 (38.2)
Q13: Avoid suffering from respiratory problems (N = 237)	7 (3.0)	37 (15.6)	101 (42.6)	92 (38.8)
Q14: Keep my health stable (N = 234)	7 (3.0)	35 (15.0)	115 (49.1)	77 (32.9)
Q15: Sleep better (N = 235)	12 (5.1)	35 (14.9)	76 (32.3)	112 (47.7)
Q16: Reduce the humidity level (N = 231)	4 (1.7)	24 (10.4)	111 (48.1)	92 (39.8)
Perceived barriers to AC				
'Does/could the following reason prevent you or limit you from using your air conditioner at home during a heat wave':				
Q17: It is too expensive to buy or run (N = 237)	85 (35.9)	113 (47.7)	20 (8.4)	19 (8.0)
Q18: It is difficult to adjust the temperature (N = 237)	79 (33.3)	146 (61.6)	10 (4.2)	2 (0.8)
Q19: It is not good for my health (N = 236)	81 (34.3)	132 (55.9)	15 (6.4)	8 (3.4)
Q20: It can make certain health problems worse (N = 237)	72 (30.8)	127 (54.3)	27 (11.5)	8 (3.4)
Q21: It prevents fresh air from getting in (N = 238)	69 (29.0)	126 (52.9)	31 (13.0)	12 (5.0)
Q22: It makes my home too cold (N = 237)	71 (30.0)	126 (53.2)	33 (13.9)	7 (3.0)
Q23: It is not comfortable (N = 238)	78 (32.8)	122 (51.3)	31 (13.0)	7 (2.9)
Q24: It makes too much noise (N = 235)	72 (30.6)	122 (51.9)	27 (11.5)	14 (6.0)
Perceived severity of heat effects on health				
'During a heat wave, if I do not protect myself from the heat':				
Q67: Respiratory difficulties caused by the heat can lead me to be hospitalized (N = 238)	0 (0.0)	44 (18.5)	88 (37.0)	106 (44.5)
Q68: Being weakened by the heat can lead me to be hospitalized (N = 238)	0 (0.0)	49 (20.6)	92 (38.7)	97 (40.8)
Q69: Dehydration caused by the heat can lead me to be hospitalized (N = 236)	2 (0.8)	49 (20.8)	93 (39.4)	92 (39.0)
Q70: Respiratory difficulties caused by the heat can provoke long-term damage to my health (N = 226)	1 (0.4)	35 (15.5)	115 (50.9)	75 (33.2)
Q71: Being weakened by the heat can provoke long-term damage to my health (N = 228)	0 (0.0)	36 (15.8)	123 (53.9)	69 (30.3)
Q72: Dehydration caused by the heat can provoke long-term damage to my health (N = 228)	0 (0.0)	41 (18.0)	117 (51.3)	70 (30.7)
Q73: Respiratory difficulties caused by the heat can lead to my death (N = 231)	6 (2.6)	74 (32.0)	95 (41.1)	56 (24.2)
Q74: Being weakened by the heat can lead to my death (N = 231)	7 (3.0)	75 (32.5)	101 (43.7)	48 (20.8)
Q75: Dehydration caused by the heat can lead to my death (N = 231)	7 (3.0)	62 (26.8)	104 (45.0)	58 (25.1)

Table I. *Continued*

Items	Strongly disagree, *n* (%)	Disagree, *n* (%)	Agree, *n* (%)	Strongly agree, *n* (%)
Perceived susceptibility to heat effects				
'Because of my state of health, if I do not protect myself from the heat':				
Q64: I am more likely to suffer from respiratory difficulties during a heat wave (N = 238)	1 (0.4)	13 (5.5)	74 (31.1)	150 (63.0)
Q65: I am more likely to become weak during a heat wave (N = 238)	0 (0.0)	17 (7.1)	85 (35.7)	136 (57.1)
Q66: I am more likely to become dehydrated during a heat wave (N = 237)	1 (0.4)	22 (9.3)	87 (36.7)	127 (53.6)
Cues to action	Not at all sensitive, *n* (%)	Not really sensitive, *n* (%)	Somewhat sensitive, *n* (%)	Very sensitive, *n* (%)
How sensitive are you to heat? (N = 237)	4 (1.7)	42 (17.7)	73 (30.8)	118 (49.8)
	Yes, *n* (%)	No, *n* (%)		
Have you heard an extreme heat warning [this summer or last summer]? (N = 237)	200 (84.4)	37 (15.6)		
Has your doctor or nurse ever told you that your health problem can make you more sensitive to the heat? (N = 237)	137 (57.8)	100 (42.2)		

[40, 41], ESEM allows for the simultaneous estimations of exploratory factor analysis (EFA) and confirmatory factor analysis models. It was deemed appropriate for use with the present data set given the need to evaluate the unknown factor structure of newly created scales and to estimate direct paths between factors in a context where small sample size precludes cross-validation. Analyses were conducted using MPlus Version 6 [42]. Default options were used as for the estimation [weighted least squares mean and variance adjusted (WLSMV)] and rotation methods (GEOMIN). The WLSMV estimator is recommended for categorical outcomes (B. O. Muthén, S. C. du Toit, D. Spisic, unpublished results). Its good performance for smaller sample sizes and computational speed has also been underscored [43]. Cues to Action was modeled as a composite variable [44]. Two measurement models using multiple indicators of latent constructs could indeed be defined. In line with classical test theory, the direction of causality could flow from construct to measure (principal factor or reflective model). Alternatively, it could also flow from measure to construct, as is the case in a composite latent variable or formative model. Given this and other criteria (indicators being not interchangeable, etc.) [45], it appeared reasonable to model the Cues to Action construct as an index produced by the observed variable (sensitivity to heat, advice from their doctor and having heard of an extreme heat warning) rather than an underlying constructs causing them.

Finally, exploratory analysis revealed a high degree of colinearity (r = 0.65) between severity and perceived susceptibility. Following a procedure used in another HBM validation study [46], perceived susceptibility was not included in the model.

Ethics

The Research Ethics Board of McGill University and those of the participating hospitals approved the study protocol. Participants signed informed consent forms after being apprised of the means used to protect their confidentiality and being assured that their responses would in no way affect their health care.

Results

Respondents' characteristics

Table II shows respondents' sociodemographic and health characteristics. Their average age was 67.8 years; 61% were male. About 16% were widowed, while the remainder were married or living with a partner (51%) or single, separated or divorced (33%). The personal income data show that about one-quarter of respondents (27%) could be considered as living in poverty, with incomes below $15 000 Canadian per year. Respondents reported a number of health problems over and above those for which they were recruited (COPD or CHF): at least 26% of respondents reported suffering from arthritis or rheumatism, insomnia, diabetes, asthma or a vision problem.

Table II *Participants' sociodemographic and health characteristics (N = 238 unless indicated)*

Characteristics	*n* (%)
Age (years) (*n* = 236)	
40–59	51 (21.6)
60–69	79 (33.5)
70–79	83 (35.2)
⩾80	23 (9.7)
Sex	
Male	145 (60.9)
Female	93 (39.1)
Marital status (*n* = 237)	
Married	122 (51.5)
Single/separated/divorced	78 (32.9)
Widowed	37 (15.6)
Living situation (*n* = 237)	
Alone	92 (38.8)
With others (spouse or other people)	145 (61.2)
Mother tongue	
French	189 (79.4)
English	36 (15.2)
Other	13 (5.5)
Annual household income (*n* = 213)	
⩽$14 999	58 (27.2)
$1500–$29 999	68 (31.9)
⩾$30 000	87 (40.8)
Years of education (*n* = 237)	
⩽6	27 (11.4)
7–12	121 (51.1)
⩾12	89 (37.6)
Health problems	
COPD or other pulmonary condition	160 (67.2)
Cardiac insufficiency	117 (49.2)
Arthritis/rheumatism	106 (44.5)
Insomnia	76 (31.9)
Diabetes	63 (26.5)
Asthma	63 (26.5)
Vision problem	62 (26.1)

AC use

Almost three-quarters (174 or 73%) of study participants reported having a home air conditioner (at least one unit in at least one room). When asked about their air conditioner use during heat waves, 31.5% reported that they occasionally or never spend time in an air-conditioned location, while 68.1% indicated doing so always or often. The average number of hours spent per 24-hour period in air-conditioned spaces during heat waves was 14.5 hours (SD = 9.4). There were few sociodemographic differences between those participants with and without home AC. *t*-tests showed no significant differences between those with ($n = 173$) and without ($n = 64$) home air conditioners were not significantly different ($p < 0.05$) in terms of gender, income, living arrangement, and physical or mental health status as measured by the SF-36 subscales. However, those with home air conditioners were marginally older than those without (68.5 versus 65.8 years, $t(234) = -1.92$, $P = 0.06$).

Structural equation model fitting

The factor loadings gave a clear interpretation of the factors in term of perceptions of severity, barriers and benefits (see Table III). Nevertheless, several items had significant cross-loadings, confirming the suitability of an ESEM approach. Only the item reflecting sensitivity to heat was a significant predictor of variability in the Cues to Action composite with items related to advice from their doctor and having heard an extreme heat warning being non-significant.

The chi-square test of exact fit emerged as statistically significant (χ^2 [277, $N = 235$] = 879.03, $P < 0.001$). Sample size independent indexes provide a more nuanced picture with the Comparative Fit

Table III. *Factor loadings for EFA with GEOMIN rotation of perception of barriers and benefits to AC use and perception of severity of health effects of heat wave items*

Items	Barriers		Benefits		Severity	
	Standard estimate	95% CI	Standard estimate	95% CI	Standard estimate	95% CI
Benefits						
Q12: Daily activities	0.27	0.21, 0.51	**0.64**	0.52, 0.76	−0.04	−0.22, 0.14
Q13: Avoid respiratory problems	0.05	−0.29, 0.38	**0.77**	0.66, 0.87	0.12	−0.12, 0.37
Q14: Health stable	0.14	−0.17, 0.46	**0.80**	0.69, 0.91	0.06	−0.16, 0.28
Q15: Sleep better	0.17	−0.08, 0.43	**0.59**	0.49, 0.70	0.10	−0.09, 0.29
Q16: Reduce humidity	0.26	0.03, 0.50	**0.60**	0.49, 0.71	0.00	−0.07, 0.08
Barriers						
Q17: Expensive	**0.57**	0.47, 0.68	−0.08	−0.22, 0.07	−0.05	−0.21, 0.11
Q18: Temperature	**0.73**	0.65, 0.81	0.01	−0.09, 0.11	−0.01	−0.12, 0.11
Q19: Not good for health	**0.85**	0.78, 0.92	0.07	−0.05, 0.20	0.08	−0.05, 0.22
Q20: Health problems	**0.78**	0.71, 0.85	0.02	−0.09, 0.13	0.09	−0.07, 0.24
Q21: Fresh air	**0.57**	0.48, 0.66	0.03	−0.10, 0.15	0.02	−0.10, 0.14
Q22: Home too cold	**0.77**	0.69, 0.85	0.18	0.06, 0.30	0.02	−0.05, 0.09
Q23: Not comfortable	**0.79**	0.69, 0.89	0.26	0.14, 0.38	−0.02	−0.09, 0.04
Q24: Too much noise	**0.50**	0.39, 0.60	0.21	0.08, 0.34	0.04	−0.09, 0.16
Severity						
Q67: Respiratory difficulties/hospitalization	−0.02	−0.10, 0.05	−0.14	−0.27, −0.00	**0.98**	0.92, 1.04
Q68: Weakened/hospitalization	0.00	−0.05, 0.06	−0.18	−0.30, −0.05	**1.02**	0.96, 1.08
Q69: Dehydration/hospitalization	−0.01	−0.07, 0.05	−0.25	−0.37, −0.12	**0.90**	0.81, 0.98
Q70: Respiratory difficulties/long-term damages	−0.04	−0.11, 0.04	**−0.67**	−0.81, −0.54	**0.99**	0.83, 1.16
Q71: Weakened/long-term damages	0.03	−0.04, 0.09	**−0.69**	−0.84, −0.54	**1.04**	0.86, 1.22
Q72: Dehydration/ long-term damages	0.05	−0.04, 0.14	**−0.56**	−0.70, −0.42	**0.97**	0.82, 1.12
Q73: Respiratory difficulties/death	**−0.47**	−0.66, −0.28	0.09	0.01, 0.16	**1.01**	0.88, 1.13
Q74: Weakened/death	**−0.51**	−0.71, −0.32	0.01	−0.02, 0.04	**1.05**	0.93, 1.16
Q75: Dehydration/death	**−0.38**	−0.55, −0.21	−0.01	−0.05, 0.03	**0.96**	0.87, 1.05

Factor loadings >0.40 are in boldface; correlations between factors: barriers–severity, 0.43; barriers–benefits, 0.30; severity–benefits, 0.46.

Index (0.955), the Tucker–Lewis Index (0.943) and the root mean squared error of approximation (0.096) reflecting acceptable to marginal fit. Although more research is needed regarding the appropriateness of such indexes for ESEM models given their greater number of estimated parameters [41], it is reasonable to conclude that the selected HBM components provide an adequate explanation of AC use (see Fig. 1). Overall, 31.6% of the variance in AC use behavior was accounted for by components of the HBM.

That is, the model showed that three components, perceived benefits, perceived barriers and cues to action, contributed significantly to AC use, in the expected directions: those who saw benefits such as allowing them to continue daily activities and to avoid respiratory problems were more likely to have a higher level of AC use. Perceptions that air conditioner use could have drawbacks such as aggravating health problems or cooling the house too much were predictive of less AC use. Cues to action were associated with higher level of AC use. Perceived severity of health problems associated with extreme heat, measured as the beliefs that heat waves could lead to hospitalization because of the respondent's health status, also did not significantly predict AC use behavior.

In an ancillary analysis, we tested a model including a direct relationship between annual household income and AC use. The estimated coefficient emerged as non-significant (results available upon request to the first author).

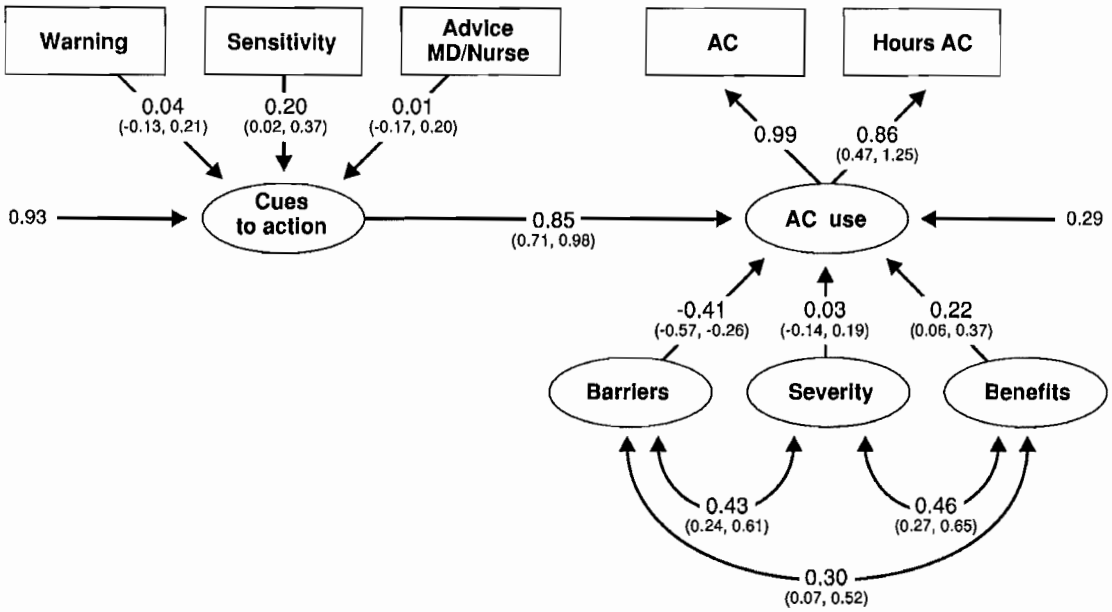

Fig. 1. Depiction of the causal model between perceived benefits, perceived barriers, perceived severity, cues to action and AC use, with standardized causal coefficients. Unstandardized residual variances for Cues to Action and AC use were constrained to 0.3. The coefficient linking AC use to AC was constrained to 1.0.

Discussion

The purpose of this study was to test the predictive power of selected components of the HBM for heat-related protective behavior among non-institutionalized middle-aged and older adults with serious chronic health conditions. Semenza *et al.* [24] estimated that more than half of the deaths related to the Chicago heat wave of 1995 'could have been prevented if each home had a working air conditioner' (p. 487). Our data partially validate the HBM by suggesting that above and beyond access to AC, beliefs in this particularly vulnerable population about AC may determine its actual level of use. About one-third of our sample of persons with CHF or COPD did not use AC regularly or did not use at all during periods of extreme heat, and those who used it, did so for, on average, just over half of each 24-hour period. ESEM showed that specific beliefs about the benefits of and drawbacks to AC, as well as cues to action were predictive of its level of use. Those who believed that using AC would help them, for example, to continue their daily activities, prevent respiratory problems and maintain their health were most likely to use it more. Similarly, those who believed that AC would bring drawbacks did not use AC as much.

While results showed a positive predictive role for cues to action, a closer look at estimates showed that only one causal indicator, sensitivity to heat, significantly contributes to the composite variable, with parameters for indicators related to advisories and advice from doctor or nurse emerging as non-significant. While this result certainly confirms the importance in the adoption of protective behavior of internal cues such as symptoms over external signals such as advisories or advice from a health professional [47], it might also reflect the difficulty of empirically identifying cues instigating action [28].

Effective communication plans and public education tools have been highlighted as key components of the public health response to extreme heat events [3]. However, current strategies such as heat advisories do not appear to be reaching their targets effectively and contribute little to the adoption of

protective behaviors [15, 24]. Although the importance of defining and locating high-risk populations, such as older adults and the chronically ill, and providing them with effective information about extreme heat has been emphasized [3, 48], the best ways to reach these vulnerable subgroups remain unclear [36]. Our data support these findings, showing that extreme heat advisories and advice from a doctor or nurse do not significantly contribute to the cues to action composite variable. Some authors have argued that communications should be improved by increasing public awareness that heat advisories are based on human health responses and by emphasizing the vulnerability aspect through their messaging [22]. This has been recommended in particular for the older adults [49]. However, as discussed by Champion and Skinner [28], it is possible that the predictive value of susceptibility may depend on the perceived level of severity so that 'a heightened state of severity is required before perceived susceptibility becomes a powerful predictor' (p. 61). This suggests that perceptions of vulnerability may be heightened most effectively when heat waves are seen as having more serious consequences on health. This hypothesis is consistent with work that has shown the potential in exploring alternative patterns of relationships for the HBM components [28], and this would certainly deserve further research. Interestingly, warning older people about health risks associated with aging and heat may have paradoxical effects: many older people do not see themselves as old or at risk [36] and those who associate aging with limitations in their lives are less likely to engage in preventive or adaptive behaviors [32, 50].

Several issues limit the interpretation of the current findings. First, although the recruitment mode allowed for the recruitment of a heterogeneous sample of non-institutionalized cardiac and COPD patients, representativeness is not guaranteed: still, the age and sex (a male majority) distribution of study participants does reflect the mix of CHF and COPD clinic patients in Montréal (according to the directors of the participating clinics), and the choice of university clinics facilitated recruitment of participants from across the socioeconomic spectrum. Second, the study is limited by its cross-sectional design. Longitudinal studies following the health belief patterns of older adults over time and before and after persuasive interventions would be invaluable in identifying the determinants of heat protective behaviors. Third, the sample size precluded the testing of alternative patterns of relationships, such as multiplicative approaches to major dimensions in the model. Further research should consider replication with larger samples and extensions with alternative causal models.

Despite its limitations, the present study is important in that it is one of the first, if not the first, to examine the usefulness of selected components of the HBM in understanding preventive behaviors related to climate change phenomena such as extreme heat. For non-institutionalized persons with existing serious health conditions, our data suggest that public health communications should include strategies aimed at modifying beliefs about specific health benefits from AC use, as well as strategies to reduce or mitigate the perceived barriers to effective action, especially among populations whose existing health problems also help to limit their mobility, restrict the social networks and isolate them [14, 49]. Such public health communications should form part of an integrated multidimensional emergency strategy [2, 34] that supports adequate citizen response [51].

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Conflict of interest statement

None declared.

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Air-conditioning and Mortality in Hot Weather

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A cohort of 72,740 persons for whom information on household air-conditioning was available was monitored for mortality via the National Death Index from April 1980 through December 1985. A total of 2,275 deaths occurred among the members of this cohort. The basic question addressed was whether persons in households with air-conditioning experienced lower death rates during hot weather than persons in households without air-conditioning. This question was examined for both central and room air-conditioning. The analysis was based on a state-by-state approach, that cross-tabulated deaths by air-conditioning status (yes or no) and average temperature during the month of death (<21.2°C (<70°F) or ≥21.2°C (≥70°F)). The Mantel-Haenszel and sign tests were used to summarize the data. For central air-conditioning versus no air-conditioning, statistically significant benefits ($p < 0.05$, Mantel-Haenszel test) were observed for the overall total, for females, for persons not in the labor force, and for persons living in fewer than six rooms. These groups had more exposure to air-conditioning. The relative risk for the total group was 0.58, implying that in hot weather, the death rate for persons who had central air-conditioning was 42 percent lower than the rate for persons who did not have air-conditioning, after confounding variables had been controlled for. For room air-conditioning versus no air-conditioning, the odds ratio for the total group was 0.96, which was not significantly different from 1.0, suggesting that no real benefit was derived from room air-conditioning. Some reasons for the lack of a demonstrable benefit for room air-conditioning are given. *Am J Epidemiol* 1992;136:106–16.

air-conditioning; demography; mortality

Excess deaths in hot weather have long been the subject of study, both in the United States and elsewhere, with some studies dating back to the last century (1–3). More recently, studies range from Gover's (4) study of weekly deaths in 86 large US cities to comprehensive studies of monthly deaths in the United States by Ellis (5) and daily deaths in 32 US standard metropolitan statistical areas by Rogot and Padgett (6). These studies generally show increased mortality during very hot weather, not only from causes directly attributable to the heat, such as heatstroke, but from causes such as heart disease, cerebrovascular disease, and respiratory disease (4–6).

Of special interest here are a retrospective case-control study by Kilbourne et al. (7) and a companion paper by Jones et al. (8) in which potential risk factors for heatstroke were studied for the heat wave in July 1980 in St. Louis and Kansas City, Missouri. Information on risk factors was obtained by questionnaire for 156 persons with heatstroke and 462 control subjects. A stepwise linear logistic regression procedure was used to identify factors significantly associated

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Abbreviation: OR, odds ratio.

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with heatstroke. Factors associated with decreased risk included the use of home air-conditioning and spending more time in air-conditioned places. This is the only study we know of that has investigated the potential benefit of air-conditioning in reducing the risk of disease.

The aim of this report is to evaluate the potential benefit of air-conditioning in reducing mortality in hot weather. In particular, during periods of hot weather, is mortality for persons in households with air-conditioning lower than for persons in households without air-conditioning? If so, can we attribute the lower mortality to air-conditioning?

MATERIALS AND METHODS

As part of the 1980 decennial census, a sample of persons and housing units was enumerated on a "long-form" questionnaire that requested information on housing characteristics and social and economic factors in addition to basic demographic information such as age, race, and sex. The item of special interest from this questionnaire is item H27:

Do you have air-conditioning?

- Yes, a central air-conditioning system
- Yes, 1 individual room unit
- Yes, 2 or more individual room units
- No

Two census cohorts from an ongoing mortality follow-up study, the National Longitudinal Mortality Study (9), were available: the April 1980 Current Population Survey (comprising some 183,000 records) and the Enumeration Sample, or E-sample, drawn from the 1980 Census (comprising approximately 250,000 records). These cohorts were matched to the census long-form files (about 45 million records), and about one sixth of the respondents were found to have completed long-form questionnaires. In all, there were 72,740 persons, excluding those in group quarters, for whom long-form records were available and who could be considered as a cohort. These persons were known to be alive in April 1980, and were eligible for mortality follow-up via the National Death Index for 5¾ years, from April 1980 through December 1985. During this period, 2,725 deaths occurred among the members of this cohort.

Ascertainment of death was carried out in two steps (9). The first step, performed at the National Center for Health Statistics, consisted of matching Census Bureau records to the National Death Index using their matching algorithm. The results of this match were reviewed in a second step at the Census Bureau using a modified Newcombe model approach to record linkage, in order to screen out false-positive matches (10). The probabilistic method used for this study is documented in reference 11; related material may be found in references 12 and 13.

The temperature data used for this study were obtained from the National Climatic Data Center. Each state is divided into as many as 10 climatologic divisions. The monthly average temperatures for each weather station within a division are collected and averaged together using equal weights to obtain the average temperature for a division. The average temperatures for all the climatologic divisions within each state are then averaged together using the proportion of the state population within each division in 1980 as weights (14). The population-based weights were chosen because they were thought to be the best representation of the temperature that a "typical person" in that state was exposed to during a given month. We defined a "hot" month as one in which the average temperature was 21.2°C (70°F) or greater and a "cold" month as one in which the average temperature was less than 21.2°C.

Persons who own air conditioners may differ in many respects from those who do not. However, since there are many socioeconomic and geographic factors, such as state of

residence, income, age of housing, number of rooms, etc., that are strongly associated with the use of air-conditioning and are also related to mortality risk, we adopted an approach designed to control for all such factors.

We assumed that the distribution of the population with air-conditioning is identical in hot months and cold months. Likewise, the distribution of the population without air-conditioning is identical in hot months and cold months.

Start with the following basic fourfold table:

Air-conditioning	Months	
	Hot	Cold
Yes	r_1	r_2
No	r_3	r_4

where r_1 is the death rate during the hot months for persons in households with air-conditioning; r_2 is the death rate during the cold months for persons in households with air-conditioning; r_3 is the death rate during the hot months for persons in households without air-conditioning; and r_4 is the death rate during the cold months for persons in households without air-conditioning. If H_0 is true, that is, no benefit is derived from air-conditioning, then

$$r_1/r_2 = r_3/r_4.$$

If a benefit is derived from air-conditioning, then r_1 is reduced and observed to be r_1', and

$$r_1'/r_2 < r_3/r_4.$$

The benefit due to air-conditioning is the difference in rates:

$$r_1 - r_1' = (r_2 r_3/r_4) - r_1'.$$

The death rates can be calculated as follows:

Air-conditioning	Average monthly population		No. of months		No. of deaths		Deaths per population per month	
	Hot months	Cold months	Hot months	Cold months	Hot months	Cold months	Hot months	Cold months
Yes	n_1	n_2	m_1	m_2	d_1	d_2	d_1/m_1n_1	d_2/m_2n_2
No	n_3	n_4	m_3	m_4	d_3	d_4	d_3/m_3n_3	d_4/m_4n_4

If H_0 is true, then

$$(d_1/m_1n_1)/(d_2/m_2n_2) = (d_3/m_3n_3)/(d_4/m_4n_4).$$

We assume that $n_1 = n_2$ and $n_3 = n_4$. Also, since $m_1 = m_3$ and $m_2 = m_4$, H_0 reduces to

$$d_1/d_2 = d_3/d_4.$$

If a benefit is derived from air-conditioning, then d_1 is reduced and observed to be d_1', and

$$d_1'/d_2 < d_3/d_4.$$

The benefit due to air-conditioning is the difference

$$\Delta = d_1 - d_1' = (d_2 d_3/d_4) - d_1'.$$

The relative benefit as a percentage is

$$100 \times \Delta/d_1 = 100 \times \Delta/(d_2d_3/d_4).$$

The relative risk for air-conditioning is

$$(d_1'/d_1) = d_1'/(d_2d_3/d_4).$$

Persons who live in households with air-conditioning may differ in many respects from those who do not. However, since the population is assumed to be identically distributed by age, race, sex, income, etc. during the hot and cold months, there is no need to adjust for confounding variables. Hot and cold months differ greatly by state (Appendix table 1), as does the use of air-conditioning (Appendix table 2), so that an analysis by state seems appropriate. Since the benefit of air-conditioning, if any, may be small, the analysis needs to take into account the exact hot and cold months for each state. To summarize the data in a state-by-state approach, the Mantel-Haenszel test (15) was used as well as a nonparametric analysis, the sign test (16), giving equal weight to each state. Room air-conditioning was evaluated as well as central air-conditioning.

For the Mantel-Haenszel test, the following basic fourfold table of observed deaths for each state was set up:

Air-conditioning	Months		
	Hot	Cold	
Yes	A	B	N_1
No	C	D	N_2
	M_1	M_2	T

where A equals deaths occurring in households that had air-conditioning during a month in which the average temperature was 21.2°C (70°F) or greater; B equals deaths occurring in households that had air-conditioning during a month in which the average temperature was less than 21.2°C (70°F); C equals deaths occurring in households that had no air-conditioning during a month in which the average temperature was 21.2°C (70°F) or greater; and D equals deaths occurring in households that had no air-conditioning during a month in which the temperature was less than 21.2°C (70°F).

A corrected chi-square with one degree of freedom and an odds ratio (OR) were calculated as follows:

$$\chi^2 = (|\sum A - \sum M_1N_1/T| - 0.5)^2/\sum[M_1M_2N_1N_2/T^2(T-1)]$$

$$OR = \sum(AD/T)/\sum(BC/T),$$

where the summation in all equations is over all states. If air-conditioning is beneficial, we would expect cell A to be smaller than expected. Consequently, odds ratios smaller than 1.0 indicate a beneficial effect for air-conditioning.

For the sign test, the number of states in which a benefit from air-conditioning was demonstrated was compared with the number showing a loss. A corrected chi-square for the sign test with one degree of freedom was calculated as follows:

$$\chi^2 = (|N_b - N/2| - 0.5)^2/N$$

where N_b is the number of states in which a benefit was demonstrated, and N is the total number of states in which either a benefit or a loss was demonstrated.

The cutpoint of 21.2°C (70°F) was chosen after preliminary work with earlier data (mortality follow-up through 1983). Cutpoints of 15.6, 18.4, 23.9, and 26.7°C (60, 65, 75, and 80°F, respectively) were also tested. The results for cutpoints of 18.4°C (65°F) and 23.9°C (75°F) were similar to those for 21.2°C (70°F) but were not as clear-cut. For the purposes of this study, we assumed that when the average temperature was 21.2°C or greater, air-conditioning would be turned on most of the time, and that when the average temperature was less than 21.2°C, air-conditioning would be turned off most of the time.

RESULTS

Table 1 summarizes the major findings for central air-conditioning. When central air-conditioning was compared with no air-conditioning, statistically significant benefits ($p < 0.05$, Mantel-Haenszel test) were observed for the overall total, females, persons not in the labor force, and persons living in fewer than six rooms. A nearly significant benefit from central air-conditioning was noted for persons aged ≥65 years ($p = 0.06$, Mantel-Haenszel test; $p = 0.10$, sign test). The odds ratio from the Mantel-Haenszel test was less than 1.0 for all groups shown, indicating some benefit from central air-conditioning, rather than a loss.

When room air-conditioning was compared with no air-conditioning, the odds ratio for the total group was 0.96, which was not significantly different from 1.0, suggesting no real benefit from room air-conditioning (table 2). The odds ratios for the subgroups shown were in all but one instance close to 1.0, some being greater than 1.0. The one exception was an odds ratio of 0.41 for persons living in 1–3 rooms. In this instance, a statistically significant benefit was attributed to room air-conditioning ($p = 0.046$, Mantel-Haenszel test; $p = 0.02$, sign test).

In tables 1 and 2, we note that the odds ratios were lower for females than males; lower for those aged 65 years or more as compared with those aged less than 65 years; lower for persons not in the labor force than

TABLE 1. Summary of findings for central air-conditioning compared with no air-conditioning, by sex, by age, by employment status, and by number of rooms in living quarters: Long-Form Follow-up Study, April 1980–December 1985

	No. of states	Total deaths	Mantel-Haenszel test		Central vs. no air-conditioning (no. of states)			Sign test p value*
			p value*	Odds ratio	Loss	Benefit	Tie	
Total	40	1,670	0.03	0.73	14	26	0	0.08
Sex								
Male	37	913	0.50	0.86	16	21	0	0.67
Female	35	618	0.02	0.58	12	23	0	0.09
Age (years)								
<65	34	564	0.65	0.87	12	21	1	0.16
≥65	38	963	0.06	0.68	13	24	1	0.10
Employment status								
In labor force	26	313	0.56	0.80	12	14	0	0.84
Not in labor force	39	1,212	0.04	0.68	13	24	2	0.10
No. of rooms in living quarters								
<6	37	863	0.04	0.64	11	25	1	0.03
1–3	14	124	0.53	0.66	5	9	0	0.42
4–5	36	669	0.18	0.70	13	21	2	0.23
≥6	38	725	0.79	0.92	17	20	1	0.74

* Two-sided probability.

TABLE 2. Summary of findings for room air-conditioning compared with no air-conditioning, by sex, by age, by employment status, and by number of rooms in living quarters: Long-Form Follow-up Study, April 1980–December 1985

	No. of states	Total deaths	Mantel-Haenszel test		Room vs. no air-conditioning (no. of states)			Sign test *p* value*
			p value*	Odds ratio	Loss	Benefit	Tie	
Total	40	1,879	0.71	0.96	22	18	0	−0.64
Sex								
Male	39	1,037	−0.85	1.05	19	20	0	1.00
Female	35	744	0.31	0.81	15	19	1	0.61
Age (years)								
<65	35	628	−0.40	1.22	17	18	0	1.00
≥65	39	1,197	0.26	0.83	16	21	2	0.67
Employment status								
In labor force	29	369	−0.78	1.10	16	12	1	−0.57
Not in labor force	40	1,426	0.55	0.91	18	20	2	0.87
No. of rooms in living quarters								
<6	40	1,090	0.67	0.92	19	20	1	1.00
1–3	19	184	0.05	0.41	4	15	0	0.02
4–5	39	841	−0.62	1.12	19	19	1	1.00
≥6	33	710	−0.96	1.01	19	13	1	−0.38

* Two-sided probability; a minus sign indicates that more states showed a loss from air-conditioning than showed a benefit.

for persons in the labor force; and lower for persons living in fewer than six rooms than for persons living in six rooms or more. This was true when central air-conditioning was compared with no air-conditioning and also when room air-conditioning was compared with no air-conditioning. For those groups showing statistically significant benefits from air-conditioning, relative risks may be calculated, as shown in table 3.

The comparison of central air-conditioning with no air-conditioning for the total group is set out in detail in table 4; the basic fourfold tables of deaths are shown for each of the 40 states in this analysis. The largest benefit, in terms of the absolute number of deaths, is shown for Florida, 51.7, which is about half the benefit of all 40 states combined, 96.5. Other states with large benefits were South Carolina (10.5), Nebraska (8.2), Missouri (7.1), New Mexico (6.0), and Illinois (5.3). In all, a benefit from central air-conditioning was found in 26 states. There were 14 states showing losses; the largest were Kansas (−3.3), New York (−3.0), Arizona (−2.4), Alabama (−2.3), and Indiana (−2.3). No particular geographic pattern was discerned here. To assess whether the benefit was greater in states with more hot months, the correlation between the benefit versus the number of hot months (average temperature ≥70°F (≥21.2°C))

TABLE 3. Relative risks for groups showing statistically significant benefits from air-conditioning: Long-Form Follow-up Study, April 1980–December 1985.

	Deaths*		
	Observed (ΣA)	Expected ($\Sigma BC/D$)	Relative risk (observed/expected)
	Central air-conditioning vs. no air-conditioning		
Total	133	229.5	0.58
Female	51	122.8	0.42
Not in labor force	94	188.9	0.50
<6 rooms	63	150.6	0.42
	Room air-conditioning vs. no air-conditioning		
1–3 rooms	19	39.1	0.41

* Observed and expected deaths for cell *A* of the basic fourfold table, i.e., for deaths during hot weather of persons with air-conditioning.

TABLE 4. Deaths by state, air-conditioning status of household (central or none) and average temperature for the month of death:* Long-Form Follow-up Study, April 1980–December 1985

State	Deaths†				Benefit‡	State	Deaths†				Benefit‡
	A	*B*	*C*	*D*	(*BC*/*D*) − *A*		*A*	*B*	*C*	*D*	(*BC*/*D*) − *A*
Alabama	4	4	3	7	−2.3	Missouri	6	25	11	21	7.1
Arizona	8	13	3	7	−2.4	Nebraska	1	23	4	10	8.2
Arkansas	5	8	2	3	0.3	Nevada	5	17	1	3	0.7
California	4	30	12	64	1.6	New Jersey	0	11	6	26	2.5
Connecticut	1	0	2	18	−1.0	New Mexico	7	13	4	4	6.0
Delaware	0	4	5	5	4.0	New York	4	5	18	91	−3.0
Florida	22	17	13	3	51.7	North Carolina	2	6	5	16	−0.1
Georgia	3	5	6	15	−1.0	North Dakota	0	2	3	43	0.1
Idaho	0	9	2	37	0.5	Ohio	1	17	16	64	3.3
Illinois	8	28	10	21	5.3	Oklahoma	1	7	2	3	3.7
Indiana	5	11	5	20	−2.3	Pennsylvania	1	12	8	66	0.5
Iowa	3	14	3	18	−0.7	Rhode Island	1	2	3	31	−0.8
Kansas	7	15	2	8	−3.3	South Carolina	0	3	7	2	10.5
Kentucky	2	7	8	14	2.0	South Dakota	1	10	5	24	1.1
Louisiana	2	5	3	6	0.5	Tennessee	3	9	2	3	3.0
Maryland and District of Columbia	3	13	5	20	0.3	Texas	15	22	6	10	−1.8
Massachusetts	0	3	2	36	0.2	Utah	2	9	3	11	0.5
Michigan	0	11	12	57	2.3	Virginia	1	10	3	16	0.9
Minnesota	1	9	3	41	−0.3	West Virginia	1	1	2	16	−0.9
Mississippi	3	2	5	5	−1.0	Wisconsin	0	8	3	34	0.7
						Total	133	420	218	899	96.5
						Relative risk = 133/(133 + 96.5) = 0.58					

* <21.2°C (<70°F) or ≥21.2°C (≥70°F).
† *A*, central air-conditioning and average temperature ≥21.2°C (≥70°F); *B*, central air-conditioning and average temperature <21.2°C (<70°F); *C*, no air-conditioning and average temperature ≥21.2°C (≥70°F); and *D*, no air-conditioning and average temperature <21.2°C (<70°F).
‡ A minus sign indicates a loss.

yielded a Spearman rank correlation coefficient of 0.03.

To compare the benefits derived from central and room air-conditioning, the same 40 states were cross-tabulated by benefit or loss from central and room air-conditioning (table 5). This yielded a moderate correlation coefficient, $\varphi = 0.45$, which was significantly greater than zero ($p < 0.05$) (16). Thus, there was a tendency for states showing either benefits or losses from central air-conditioning also to show benefits or losses from room air-conditioning.

A number of other variables, in addition to those shown in tables 1 and 2, were investigated. These included family income, age of housing, and standard metropolitan statistical area status. No clear-cut patterns emerged from these analyses.

TABLE 5. Distribution of states by loss or benefit from central air-conditioning versus loss or benefit from room air-conditioning:* Long-Form Follow-up Study, April 1980–December 1985

Central air-conditioning	Room air-conditioning		Total
	Loss	Benefit	
Loss	12	2	14
Benefit	10	16	26
Total	22	18	40

* Correlation coefficient $\varphi = 0.45$.

In addition, deaths were divided by underlying cause of death into cardiovascular and noncardiovascular, which were analyzed separately. No clear-cut differences between cardiovascular and noncardiovascular deaths emerged. In general, for the variables

not seen in tables 1 and 2, when the data were subdivided, the odds ratios seen were invariably less than 1.0 for central air-conditioning compared with no air-conditioning, but were not statistically significant; on the other hand, when room air-conditioning was compared with no air-conditioning, the odds ratios were usually close to 1.0, and sometimes greater than 1.0 but without statistical significance.

Equivalent statistical analyses can be done by replacing the Mantel-Haenszel test with a logistic model. When this was done, the odds ratios so calculated confirmed the results previously obtained from the Mantel-Haenszel test.

DISCUSSION

Overall, a clear benefit was demonstrated for central air-conditioning but none was found for room air-conditioning except for persons living in 1–3 rooms. The death rate in hot weather for persons in households with central air-conditioning was much lower than that for persons in households without air-conditioning, and this lower rate may be attributed to air-conditioning. The relative risk was 0.58, implying that during hot weather, the death rate for persons with central air-conditioning was 42 percent lower than the death rate for persons without air-conditioning, after confounding variables were controlled for. This benefit of 42 percent was not shared by all the states, although benefits were seen in 26 of the 40 states in the analysis. No particular geographic pattern was seen, except that Florida contributed the largest benefit—51.7 of the 96.5 lives saved. (Without Florida, the relative risk would increase from 0.58 to 0.71 and the chance probabilities from the Mantel-Haenszel and sign tests would increase to 8 percent and 11 percent, respectively).

In interpreting these findings, two possible limitations of these data must be addressed as a cautionary note: the possibly incomplete ascertainment of death and the validity of the basic assumption that the population is the same during the hot months as during the cold months. With respect to the latter assumption, we examined the seasonal population shifts for Florida, the state with probably the most extreme shift. We assessed the effect in table 4, in which central air-conditioning is compared with no air-conditioning. The underlying death rates can be calculated as described above with the data for Florida residents reported in the Appendix tables. By assuming the same population during the hot and cold months, we obtain a relative risk for air-conditioning for Florida residents of 0.298. The proportion of all Florida residents who live in Florida for slightly more than 6 of the cold months and live in another state during the hot months is not known. If we assume this proportion to be as high as 5 or even 10 percent, we find that the corrected relative risks are only slightly higher than 0.298. The population shift for Florida is clearly not large enough to affect the study findings materially.

The overall death rate in our study is lower than that for the general population of the United States. This has been pointed out in previous reports (9, 17), and appears to be so for two reasons: 1) the study population does not include persons in nursing homes or other institutions for whom death rates tend to be high; and 2) deaths may be missed in the matching procedure used in the National Death Index.

With respect to missed deaths, there is no compelling reason to believe that the missed deaths are more likely to have occurred among persons living in households with central air-conditioning and during hot weather. The number of deaths missed in the National Death Index matching procedure is not known, but is thought to be relatively small, on the order of 5–10 percent.

It was noted earlier that odds ratios were lower, indicating greater benefits, for women as compared with men, for older persons as compared with younger persons, and for persons not in the labor force as compared with persons in the labor force. These comparisons all point to the percentage of time spent indoors as a critical factor. The more time spent indoors, the greater the exposure

to air-conditioning if it is available in the home. Thus, the more time spent indoors at home, the more closely our observational study approximates an ideal situation in which, for example, during hot weather, persons in cell *A* of the basic fourfold table are exposed 100% of the time to air-conditioning, while those in cell *C* are exposed none of the time to air-conditioning. If there is, in fact, a real benefit from air-conditioning, it will be detected more easily in the ideal situation, since this offers the greatest contrast. It is plausible that women spend more time indoors than men, that older persons are indoors more than younger persons, and that persons who are not in the labor force are indoors more than persons who are. If this is so, the differential benefits observed above would be expected.

It should also be pointed out that persons with no air-conditioning at home may nevertheless work in an air-conditioned office or drive in an air-conditioned car. Thus, the comparison of "central versus none" may in reality be "central versus some." The latter is more likely to be the case, for example, for persons in the labor force than for persons who are not in the labor force.

For room air-conditioning versus no air-conditioning, the Mantel-Haenszel odds ratio was 0.96, which was not significantly different from 1.0. Thus, overall, no real benefit for room air-conditioning was seen, in spite of the strong benefit for central air-conditioning. If this benefit is on the order of 42 percent (cited earlier), we should expect a more modest benefit—much less than 42 percent—for room air-conditioning, since only a fraction of the house is protected. The extent of the benefit should depend on the number of rooms in the house, since this would affect the exposure time in air-conditioned or non–air-conditioned space. The benefit to be expected here might be on the order of only 10–20 percent. It would be very difficult to detect such a small benefit without very large numbers.

One exception stands out: for persons living in 1–3 rooms, a substantial benefit was seen for room air-conditioning as compared with no air-conditioning. In this particular comparison, one or more room air conditioners for 1–3 rooms may be seen as nearly equivalent to central air-conditioning.

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APPENDIX TABLE 1. Number of months in the study period (April 1980–December 1985) in which the average temperature was <21.2°C (<70°F) or ≥21.2°C (≥70°F), for each state*

State	No. of months at		State	No. of months at	
	<21.2°C (<70°F)	≥21.2°C (≥70°F)		<21.2°C (<70°F)	≥21.2°C (≥70°F)
Alabama	43	26	Montana	68	1
Alaska†	69	0	Nebraska	54	15
Arizona	40	29	Nevada	47	22
Arkansas	44	25	New Hampshire	69	0
California	54	15	New Jersey	56	13
Colorado	67	2	New Mexico	52	17
Connecticut	58	11	New York	58	11
Delaware	52	17	North Carolina	48	21
Florida	26	43	North Dakota	63	6
Georgia	44	25	Ohio	58	11
Hawaii‡	0	69	Oklahoma	44	25
Idaho	64	5	Oregon	69	0
Illinois	56	13	Pennsylvania	60	9
Indiana	55	14	Rhode Island	60	9
Iowa	54	15	South Carolina	43	26
Kansas	50	19	South Dakota	58	11
Kentucky	51	18	Tennessee	49	20
Louisiana	36	33	Texas	38	31
Maine	69	0	Utah	58	11
Maryland§	53	16	Vermont	69	0
Massachusetts	60	9	Virginia	50	19
Michigan	60	9	Washington	69	0
Minnesota	64	5	West Virginia	56	13
Mississippi	41	28	Wisconsin	63	6
Missouri	52	17	Wyoming	68	1

* Temperature data obtained from the National Climatic Data Center, Asheville, North Carolina.
† Data are for Anchorage.
‡ Data are for Honolulu.
§ Washington, DC, is included as part of Maryland in these Weather Bureau statistics.

APPENDIX TABLE 2. Number of persons* by state and air-conditioning status of household: Long-Form Follow-up Study, April 1980–December 1985

State	Air-conditioning in household				Total	State	Air-conditioning in household				Total
	Central	1 room	≥2 rooms	None			Central	1 room	≥2 rooms	None	
Alabama	414	287	112	323	1,136	Montana	48	97	17	785	947
Alaska	3	0	0	915	918	Nebraska	587	330	44	302	1,263
Arizona	471	55	12	245	783	Nevada	644	96	11	186	937
Arkansas	393	262	45	309	1,009	New Hampshire	12	173	21	732	938
California	1,099	582	153	2,535	4,369	New Jersey	362	393	392	562	1,709
Colorado	154	99	15	696	964	New Mexico	362	134	21	314	831
Connecticut	54	143	146	460	803	New York	275	600	631	2,228	3,734
Delaware	171	129	117	226	643	North Carolina	347	252	97	449	1,145
District of Columbia	132	50	107	173	462	North Dakota	203	311	59	1,057	1,630
Florida	899	231	213	246	1,589	Ohio	643	626	230	1,822	3,321
Georgia	419	177	82	366	1,044	Oklahoma	449	276	119	191	1,035
Hawaii	14	82	28	594	718	Oregon	92	126	17	790	1,025
Idaho	177	126	21	1,023	1,347	Pennsylvania	290	448	330	1,627	2,695
Illinois	1,088	628	287	924	2,927	Rhode Island	31	145	105	460	741
Indiana	440	366	160	831	1,797	South Carolina	237	149	66	203	655
Iowa	427	350	73	354	1,204	South Dakota	364	461	39	678	1,542
Kansas	648	328	61	298	1,335	Tennessee	349	277	75	164	865
Kentucky	469	352	128	451	1,400	Texas	1,542	491	367	468	2,868
Louisiana	411	150	184	160	905	Utah	463	188	27	609	1,287
Maine	2	96	19	1,081	1,198	Vermont	2	88	10	1,257	1,357
Maryland	369	204	131	348	1,052	Virginia	469	166	130	326	1,091
Massachusetts	55	321	125	859	1,360	Washington	71	57	20	898	1,046
Michigan	322	396	90	2,007	2,815	West Virginia	138	229	95	644	1,106
Minnesota	328	343	77	1,030	1,778	Wisconsin	205	325	90	1,083	1,703
Mississippi	298	193	121	257	869	Wyoming	62	99	4	723	888
Missouri	783	442	188	543	1,956	United States	18,287	12,929	5,712	35,812	72,740

* Excludes persons living in group quarters.

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HEAT-RELATED DEATHS DURING THE JULY 1995 HEAT WAVE IN CHICAGO

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ABSTRACT

Background During a record-setting heat wave in Chicago in July 1995, there were at least 700 excess deaths, most of which were classified as heat-related. We sought to determine who was at greatest risk for heat-related death.

Methods We conducted a case–control study in Chicago to identify risk factors associated with heat-related death and death from cardiovascular causes from July 14 through July 17, 1995. Beginning on July 21, we interviewed 339 relatives, neighbors, or friends of those who died and 339 controls matched to the case subjects according to neighborhood and age.

Results The risk of heat-related death was increased for people with known medical problems who were confined to bed (odds ratio as compared with those who were not confined to bed, 5.5) or who were unable to care for themselves (odds ratio, 4.1). Also at increased risk were those who did not leave home each day (odds ratio, 6.7), who lived alone (odds ratio, 2.3), or who lived on the top floor of a building (odds ratio, 4.7). Having social contacts such as group activities or friends in the area was protective. In a multivariate analysis, the strongest risk factors for heat-related death were being confined to bed (odds ratio, 8.2) and living alone (odds ratio, 2.3); the risk of death was reduced for people with working air conditioners (odds ratio, 0.3) and those with access to transportation (odds ratio, 0.3). Deaths classified as due to cardiovascular causes had risk factors similar to those for heat-related death.

Conclusions In this study of the 1995 Chicago heat wave, those at greatest risk of dying from the heat were people with medical illnesses who were socially isolated and did not have access to air conditioning. In future heat emergencies, interventions directed to such persons should reduce deaths related to the heat. (N Engl J Med 1996;335:84-90.)

HOT summer weather cannot be prevented; however, morbidity and mortality related to summer heat can be reduced.[1] Previous research shows that the excess mortality associated with sustained hot weather affects predominantly high-risk populations living in urban areas where there has not been a heat wave for several years.[2-7] Simple measures such as increasing fluid intake and gaining access to air conditioning can reduce heat-related mortality.[1,8] However, implementing preventive strategies targeted to groups at risk has proved to be difficult, and many preventable deaths continue to occur throughout the United States during the summer months.[9]

From July 12 through July 16, 1995, in Chicago, the maximal and minimal temperatures reached unprecedented highs, and the high temperatures were accompanied by extremes of relative humidity (Fig. 1).[10] Within a few days of the onset of the heat spell, the Cook County Medical Examiner's Office reported a sharp increase in the number of heat-related deaths.[11]

We conducted a case–control study to determine risk factors for death due to cardiovascular causes or related to the heat. Our main objective was to identify effective public health strategies for reaching people at risk and preventing deaths in future heat waves.

METHODS

Study Design and Case Definition

We conducted a case–control study from July 21 through August 18, 1995. Eligible case subjects were persons older than 24 years of age who died in Chicago from July 14 through July 17, for whom the cause of death listed on the death certificate met one of three criteria: heat was listed as the immediate or underlying cause of death, with no reference to cardiovascular disease; cardiovascular disease was listed as the primary cause of death, with no reference to heat; or cardiovascular disease was listed as the primary cause and heat as a contributing cause of death. We included deaths due to cardiovascular causes in the case definition because previous studies had demonstrated an excess of deaths from cardiovascular disease during periods of high heat.[1,5]

In order to avoid the delay that might be involved in obtaining the death certificates of all eligible subjects, all death certificates issued in July 1995 were collected by the Vital Statistics Division of the Department of Public Health in Chicago and assigned temporary identification numbers. From a review of those death certificates, we identified 680 potential subjects who met our case definition; we then selected a random sample from each cause-of-death category, with stratification according to age, race, and date of death (Table 1). We chose the sample sizes for each category to give the study a statistical power of 80 percent for detecting an odds ratio of 2.0 among subjects with any predictive factor as

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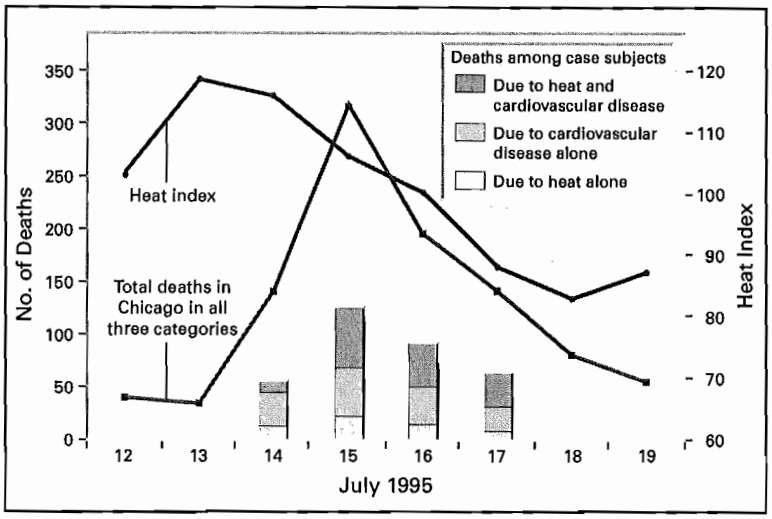

Figure 1. Heat Index, Total Deaths, and Deaths of Case Subjects Due to Heat, Cardiovascular Disease, and Heat and Cardiovascular Disease Combined in Chicago, July 12 through July 19, 1995.

The heat index, or apparent temperature, is a function of the temperature in degrees Fahrenheit and the relative humidity. It provides a measure of the evaporative and radiant transfer of heat between a typical human and the environment. Total numbers of deaths in Chicago with cardiovascular disease or heat as a primary or secondary cause were obtained from the Illinois Department of Public Health. The bars represent case subjects included in the analysis, according to cause of death.

compared with subjects without it, assuming that 18 percent of controls were exposed to any given risk factor, at a significance level of 0.05. We visited the residences of 420 potential case subjects and interviewed family members, neighbors, or friends of 372 of them (89 percent).

We identified one neighborhood control for each case subject, using a standardized search procedure; staff members began at the door of the subject's home and moved in a direction determined by coin tossing. A control of the same age was sought at the next residence. If no suitable control was identified at the buildings visited in one direction, the search was begun again from the residence of the case subject with the staff member moving in the opposite direction. The search was continued on the next floor or at the next apartment building, if necessary, with direction determined by coin tossing, until an appropriate control was found. A similar procedure was used for single-family homes. All searches were extended to the street, entire block, or neighborhood until an appropriate control was found. Controls were matched with the case subjects for age within 5 years, except for case subjects 80 or older, for whom the range was expanded to 10 years. Of 636 attempts to recruit controls, 59.4 percent led to interviews; in 14.8 percent of the attempts no one was at home; in 17.5 percent the persons identified did not match the case subjects for age; and 8.3 percent of potential controls declined to participate.

Using a uniform questionnaire, we obtained information by interviewing controls and surrogate respondents for the case subjects, such as family members, friends, or neighbors who were encountered when staff members visited the address on the death certificate. Additional information was obtained from the death certificate, police reports, and phone calls to the case subjects' next of kin. Information on the case subjects' living conditions was collected by the inspection of residences. Interviews were conducted by staff members of the Centers for Disease Control and Prevention and by trained volunteers recruited by the Chicago Department of Public Health and the Office of the Mayor.

Statistical Analysis

We used matched-pairs analysis to estimate odds ratios and confidence intervals for each potential risk factor and cause-of-death category.[12,13] For each risk factor, we calculated a summary odds ratio that combined the three odds ratios for the cause-of-death categories. This summary odds ratio was the weighted average of the three cause-specific odds ratios, with weights calculated from the corresponding sampling fraction and the relative frequency of each cause of death. We estimated the variance for the summary measure by using the delta method[14] and treating the weights as constant. In univariate analysis of data on the total population of study subjects, we used 160 potential risk factors derived from the questionnaire.

Variables that were significant in the crude, univariate analysis were entered into a conditional, stepwise logistic-regression model. The final multivariate model contained the variables that were significant predictors of death in one or more cause-of-death categories. Summary odds ratios were calculated with use of the same weights as in the crude analysis. A summary generalized impact fraction was calculated from the summary odds ratio and the weighted numbers of case subjects exposed or control subjects not exposed to a particular factor, as a measure of the impact of these factors and their relevance for public health.[13] Assuming that factors that increase risk are causally related to death, the calculation estimates the percentage of deaths attributable to a particular factor. For factors that decrease risk, the generalized impact fraction estimates the number of deaths attributable to the absence of the factor in some proportion of the population.

TABLE 1. SELECTION AND CHARACTERISTICS OF PERSONS WHO DIED OF HEAT-RELATED OR CARDIOVASCULAR CAUSES IN CHICAGO FROM JULY 14 THROUGH JULY 17, 1995, AND THEIR MATCHED CONTROLS, ACCORDING TO CAUSE OF DEATH.

VARIABLE	CAUSE OF DEATH						TOTAL	
	HEAT		HEAT AND CARDIOVASCULAR DISEASE		CARDIOVASCULAR DISEASE			
	Case Subjects	Controls	Case Subjects	Controls	Case Subjects	Controls	Case Subjects	Controls
	number (percent)							
Category								
Eligible subjects	84	—	353	—	243	—	680	—
Included in sample	80 (95)	—	201 (57)	—	192 (79)	—	473 (70)	—
Interviewed	62 (78)	65	157 (78)	162	153 (80)	151	372 (79)	378
Included in analysis	60	60	144	144	135	135	339	339
Characteristic								
Age (yr)								
<76	37 (62)	43 (72)	83 (58)	100 (69)	63 (47)	87 (64)	183 (54)	230 (68)
≥76	23 (38)	17 (28)	61 (42)	44 (31)	72 (53)	48 (36)	156 (46)	109 (32)
Race*								
Black	24 (40)	28 (47)	69 (48)	72 (51)	58 (43)	59 (44)	151 (45)	159 (47)
White	35 (58)	30 (50)	74 (51)	65 (46)	76 (56)	72 (54)	185 (55)	167 (50)
Other	1 (2)	2 (3)	1 (1)	5 (4)	1 (1)	2 (2)	3 (1)	9 (3)
Hispanic ethnic background	1 (2)	2 (3)	9 (6)	15 (10)	8 (6)	9 (7)	18 (5)	26 (8)
Sex								
Female	27 (45)	32 (53)	66 (46)	73 (51)	67 (50)	76 (56)	160 (47)	181 (53)
Male	33 (55)	28 (47)	78 (54)	71 (49)	68 (50)	59 (44)	179 (53)	158 (47)

*Missing data for the control groups are due to incomplete questionnaires.

RESULTS

Subjects

Of 750 persons — both case subjects and controls — for whom we had completed questionnaires, 678 (90 percent), or 339 matched pairs, were included in the analysis (Fig. 1 and Table 1). Case subjects whose surrogates were successfully surveyed were similar in terms of age, race, ethnic background, and sex to those whose surrogates were not surveyed. Table 1 shows selected demographic variables for case subjects and controls according to cause-of-death category. For the initial group of 680 eligible case subjects, the median age was 76 years. The proportions of black and white subjects were approximately equal among the case subjects and controls, and case subjects were generally older than controls in each cause-of-death category; the age difference was greatest between persons who died from cardiovascular causes and their controls.

Living Conditions and Social Contacts

There were fewer deaths among people who had a working air conditioner (odds ratio, 0.2; 95 percent confidence interval, 0.2 to 0.4) or had access to an air-conditioned lobby if the subject lived in an apartment building (odds ratio, 0.2; 95 percent confidence interval, 0.1 to 0.5) than among those who did not (Table 2). Visiting an air-conditioned place during the heat wave, other than the subject's residence, was associated with lower mortality (odds ratio, 0.3; 95 percent confidence interval, 0.2 to 0.5). We did not detect any reduction in mortality in association with the use of electric fans (data not shown).

Living alone, as compared with living with others, was associated with a doubling of the risk of death during the hot weather (odds ratio, 2.3; 95 percent confidence interval, 1.4 to 3.5). More than half the case subjects (52 percent) lived on the top floor of a building, as compared with less than a third of the controls (32 percent) ($P<0.05$). Of the case subjects, 57 percent lived in apartment houses, as compared with 47 percent of the controls, and 58 percent of the case subjects lived in buildings with flat roofs, as compared with 51 percent of the controls ($P<0.05$).

The case subjects lived in homes with fewer rooms, on average, than did the controls. In addition, case subjects were less likely to leave home frequently, to have friends in Chicago, to have pets, and to participate in group activities than were controls.

Medical Factors

Preexisting medical conditions were associated with an increased risk of death during the heat wave

TABLE 2. LIVING CONDITIONS AND TYPES OF SOCIAL CONTACTS AMONG 339 MATCHED PAIRS OF CASE SUBJECTS AND CONTROLS.*

VARIABLE	CASE SUBJECTS	CONTROLS	ODDS RATIO (95% CI)†
	no. (%)		
Living conditions			
Had working air conditioner in home	81 (25)	170 (53)	0.2 (0.2–0.4)
Had access to air-conditioned lobby	28 (10)	54 (20)	0.2 (0.1–0.5)
Visited cooling shelters	14 (5)	22 (7)	0.5 (0.3–1.2)
Visited other air-conditioned places	67 (22)	130 (43)	0.3 (0.2–0.5)
Lived alone	156 (46)	112 (33)	2.3 (1.4–3.5)
Lived on the top floor‡	83 (52)	51 (32)	4.7 (1.7–12.8)
Type of residence			
Single-family home or duplex§	129 (39)	165 (50)	1.0
Apartment building	185 (57)	155 (47)	2.5 (1.5–4.2)
Other kind of building	13 (4)	7 (2)	8.1 (1.4–45.8)
Number of rooms			
1 or 2	65 (20)	53 (16)	3.4 (1.5–7.9)
3 or 4	87 (27)	72 (22)	2.8 (1.4–5.3)
5 or 6	135 (41)	134 (41)	1.6 (0.9–2.7)
>6§	40 (12)	68 (21)	1.0
Lived in building with flat roof	192 (58)	167 (51)	2.0 (1.2–3.2)
Social contact			
Participated in group activities (clubs, support groups, church)	140 (46)	167 (55)	0.7 (0.5–0.9)
Had access to transportation (car, bus, or train)	262 (79)	303 (92)	0.4 (0.2–0.6)
Had friends in Chicago	288 (90)	312 (97)	0.3 (0.1–0.6)
Did not leave home¶	75 (27)	19 (7)	6.7 (3.0–15.0)
Had pet in home	78 (24)	99 (30)	0.6 (0.4–0.9)

*For each variable, the denominator is based on the number of pairs with no missing data.

†Odds ratios are calculated as the risk of death among subjects with the characteristic in question, as compared with those without it, unless otherwise specified. CI denotes confidence interval.

‡The reference category was subjects who lived below the top floor of an apartment building.

§Reference category.

¶The reference category was those who left home at least once a week.

(Table 3). Confinement to bed was the strongest predictor of death; 16 percent of case subjects were bedridden, as compared with only 4 percent of controls (odds ratio, 5.5). The need for assistance from visiting nurses was associated with a mortality rate approximately six times higher than that among people who did not receive such assistance (odds ratio, 6.2); housekeepers, home health aides, and Meals on Wheels programs were also used by subjects at increased risk (odds ratios, 2.5 to 2.7). The city of Chicago sent social-service workers to visit people at risk and explain the dangers of hot weather; a decreased risk of death was found among the people they contacted (odds ratio, 0.7).

Persons whose deaths were listed on the death certificates as due to cardiovascular causes tended to have a higher frequency of preexisting medical conditions than those whose death certificates did not list cardiovascular disease (Table 4). Other risk factors among persons whose deaths were attributed to cardiovascular causes were similar in direction to those among persons for whom heat was either the primary or a contributing cause of death. Having a working air conditioner was associated with an 80 percent reduction in the risk of death due to heat and cardiovascular disease and a 66 percent reduction in mortality due to cardiovascular disease (odds ratio, 0.3; 95 percent confidence interval, 0.2 to 0.6).

Results of Multivariate Analyses

In multivariate analyses, the strongest protective factor was having a working air conditioner in the home (odds ratio, 0.3; 95 percent confidence interval, 0.2 to 0.6) (Table 5). We estimate that more than 50 percent of the deaths related to the heat wave could have been prevented if each home had had a working air conditioner (generalized impact fraction, 50.2 percent). Also effective in reducing the risk of heat-related death were visiting an air-conditioned place (odds ratio, 0.5; 95 percent confidence interval, 0.3 to 0.9) and having access to transportation (odds ratio, 0.3; 95 percent confidence interval, 0.1 to 0.5). Confinement to bed was correlated with the inability to care for oneself and was a strong risk factor for heat-related death. Living

TABLE 3. MEDICAL CONDITIONS AND CONTACTS WITH HEALTH CARE PROVIDERS AMONG 339 MATCHED PAIRS OF CASE SUBJECTS AND CONTROLS.*

VARIABLE	CASE SUBJECTS	CONTROLS	ODDS RATIO (95% CI)†
	no. (%)		
Medical conditions			
Confined to bed	51 (16)	13 (4)	5.5 (2.5–12.1)
Unable to care for self	77 (23)	29 (9)	4.1 (2.0–8.5)
Mental problem	52 (20)	23 (9)	3.5 (1.7–7.3)
Heart condition	92 (39)	46 (19)	2.3 (1.5–3.6)
Pulmonary condition	30 (13)	14 (6)	2.2 (1.0–4.9)
Body-mass index‡			
<24	131 (45)	89 (30)	1.4 (1.0–2.2)
24–27§	81 (28)	82 (28)	1.0
≥28	81 (28)	122 (42)	0.6 (0.4–1.0)
Contact with health care providers			
Visited by nurses	62 (20)	16 (5)	6.2 (2.9–13.4)
Contacted by city workers during heat wave	25 (9)	43 (16)	0.7 (0.3–1.7)
Used housekeeper or home health aide	54 (17)	27 (9)	2.7 (1.5–4.8)
Used Meals on Wheels	22 (7)	12 (4)	2.5 (1.0–6.5)

*For each variable, the denominator is based on the number of pairs with no missing data.

†Odds ratios are calculated as the risk of death among subjects with the characteristic in question, as compared with those without it, unless otherwise specified. CI denotes confidence interval.

‡The weight in kilograms divided by the square of the height in meters.

§Reference category.

alone, possibly indicating fewer contacts with family and friends and social isolation, was also associated with increased mortality (odds ratio, 2.3; 95 percent confidence interval, 1.2 to 4.4).

Other Factors Correlated with Risk

In an effort to identify practical intervention strategies, we attempted to determine the most effective means of communication with people at risk by considering information provided by the controls in interviews. We analyzed factors associated with a lack of awareness of the potential danger of sustained heat, since the univariate analysis had indicated that increased awareness (indicated by contact with city workers) was protective. The controls who listened to the radio were more likely than others to be aware of the health risks associated with hot weather (odds ratio, 2.1; 95 percent confidence interval, 0.8 to 5.6); the same was true of the controls who read the newspaper (odds ratio, 2.5; 95 percent confidence interval, 1.2 to 5.3). Virtually all controls watched television; thus, its contribution to their awareness of heat-related dangers could not be evaluated.

We also used information supplied by the controls to evaluate factors associated with the lack of air conditioning. Residents of apartment buildings were particularly likely to lack air conditioning (odds ratio, 3.4; 95 percent confidence interval, 2.1 to 5.4), as were subjects who lived in single-room-occupancy or other hotels (odds ratio, 6.2; 95 percent confidence interval, 0.6 to 61.4), as compared with people living in single-family houses. People were more likely to change their daily routines during hot weather — for example, by drinking extra fluids or taking extra baths — if they lived with at least one other person (odds ratio, 2.1; 95 percent confidence interval, 1.3 to 3.4) or if they had a pet (odds ratio, 2.1; 95 percent confidence interval, 1.3 to 3.5).

TABLE 4. SELECTED FACTORS ASSOCIATED WITH THE RISK OF HEAT-RELATED DEATH, ACCORDING TO CAUSE OF DEATH.

VARIABLE	CAUSE OF DEATH		
	HEAT	HEAT AND CARDIOVASCULAR DISEASE	CARDIOVASCULAR DISEASE
No. of case–control pairs	60	144	135
	odds ratio (95% confidence interval)*		
Heart condition	1.8 (0.6–5.4)	1.5 (0.8–2.8)	4.5 (2.3–8.9)
Mental problem	3.0 (0.8–11.1)	2.6 (1.1–6.2)	5.0 (1.4–17.3)
Confined to bed	2.0 (0.2–22.1)	4.3 (1.4–12.6)	9.0 (2.7–29.7)
Visited by nurses	10.0 (1.2–78.1)	3.8 (1.4–10.2)	8.7 (2.6–28.6)
Unable to care for self	9.0 (1.1–71.0)	1.9 (0.9–4.1)	6.2 (2.9–14.6)
Visited other air-conditioned places	0.1 (0.04–0.5)	0.4 (0.2–0.7)	0.4 (0.2–0.8)
Had working air conditioner	0.2 (0.1–0.6)	0.2 (0.1–0.4)	0.3 (0.2–0.6)
Lived alone	5.7 (1.7–19.3)	2.2 (1.2–3.9)	1.5 (0.9–2.6)
Did not leave home	1.8 (0.5–6.0)	7.3 (2.2–24.5)	7.8 (3.1–19.8)

*Odds ratios are calculated as the risk of death among subjects with the characteristic in question, as compared with those without it.

TABLE 5. ASSOCIATION OF RISK FACTORS WITH HEAT-RELATED DEATH IN THE WEIGHTED MULTIVARIATE ANALYSIS.

VARIABLE	CASE SUBJECTS (N=339)	CONTROLS (N=339)	ODDS RATIO (95% CI)*	GIF (%)†
	no. (%)			
Had working air conditioner in home	96 (28)	170 (50)	0.3 (0.2–0.6)	50.2
Visited other air-conditioned places	103 (30)	130 (38)	0.5 (0.3–0.9)	39.5
Had access to transportation	270 (80)	303 (89)	0.3 (0.1–0.5)	16.3
Confined to bed	51 (15)	13 (4)	8.2 (3.1–22.0)	13.7
Lived alone	156 (46)	112 (33)	2.3 (1.2–4.4)	27.1

*Odds ratios are calculated as the risk of death among subjects with the characteristic in question, as compared with those without it. CI denotes confidence interval.

†GIF denotes generalized impact fraction, an estimate of the percentage of deaths attributable to a particular factor or the absence of a particular factor.

DISCUSSION

The people who are at greatest risk for death due to heat or to cardiovascular causes include those in frail health, such as elderly persons, and those who are socially isolated. People at risk were often in need of help from visiting nurses, housekeepers, or Meals on Wheels programs — an association that could have implications for preventive public health programs. Living conditions, including the type of building, the floor level, and the number of rooms, were also found to be important determinants of risk. The presence of air conditioning was inversely associated with mortality from both heat and cardiovascular causes.

Limitations

As is true in other studies that rely on information provided by surrogate respondents (in this case, family members, neighbors, or friends of people who had died), our results may underestimate the risk associated with social isolation, since people with few social contacts (and hence no identifiable surrogate) were excluded from the study. We did not find any demographic differences between the case subjects for whom surrogate respondents were available and those for whom they were not; however, we have no

details of the lifestyles of the case subjects without surrogates. Nonetheless, even after the exclusion of this most isolated segment of the population, we found a significant association between risk and variables measuring the degree of social isolation. Similarly, subjects interviewed personally and surrogate respondents are not equally credible, particularly in the case of socially isolated persons. Therefore, information concerning the case subjects' personal behavior and awareness and knowledge of health risks was excluded.

A limitation inherent in the use of death certificates is the potential misclassification of causes of death.[15-17] Almost all death certificates listing heat as a cause of death were signed by personnel from the Medical Examiner's Office rather than by private health care providers. Despite the possibility of misclassification, we found that risk factors overlapped strongly between persons whose deaths were listed as related to heat and those whose deaths were listed as due solely to cardiovascular disease.

Strategies for Prevention

Home health care workers, friends, and the media can be effective in communicating health-protection messages directly to high-risk groups. Some of the people at highest risk of death during a heat wave are those who, as a consequence of preexisting illness, have ongoing connections to community services — through visiting nurses or Meals on Wheels personnel, for example. During periods of hot weather, these and similar public health programs provide an ideal opportunity for intervention. Media coverage of appropriate preventive measures can also be effective in increasing the public's awareness of the dangers of heat. Successful messages should encourage the modification of routine behavior in the direction of prevention.

Certain housing conditions may also entail a substantial risk of heat-related death. Living in an apartment was associated with increased risk, particularly if no air conditioner was available in the lobby. Socioeconomic differences that manifest themselves in housing may confound these findings to some extent; for example, the lack of a working air conditioner was more common among inhabitants of apartment buildings than among inhabitants of private homes. This information points to the importance of directing outreach efforts toward apartment dwellers.

Previous research has yielded conflicting results regarding the effects of electric fans in hot weather[1,18-21] (and Steadman RG: personal communication). In our study, we did not find any evidence that the use of fans was protective, nor did we determine that any level of use of electric fans was associated with increased mortality. Interpretation of the data on the use of fans is complicated by the need to take into account specific environmental factors (for example, whether the fan is used in a room with an open or a closed window) and the health status of individual subjects. The effectiveness of fans in preventing death during periods of high heat and humidity remains a matter of controversy and deserves further attention; nonetheless, the potential preventive value and effectiveness of electric fans seem minimal as compared with those of other clearly beneficial factors, such as increasing use of air conditioning.

The preexisting medical conditions of individual Chicagoans greatly affected how well they were able to deal with last summer's heat wave. Although obesity is usually associated with a decreased ability to acclimate to heat,[22,23] we did not find an association between mortality and a body-mass index (defined as the weight in kilograms divided by the square of the height in meters) above the normal range. We did find that people with a body-mass index below the normal range had increased risk. For the elderly, a body-mass index lower than 24 may indicate a debilitating illness or poor nutritional status. In all three cause-of-death categories, subjects with diagnosed heart or kidney disease or mental illness had an increased risk of death. Other researchers have found that neuroleptic drugs can interfere with the physiologic response to extremes of temperature and therefore increase the risk of heat-related effects on health.[1,24] We found that of the 59 case subjects who were at increased risk because of preexisting mental problems, 2 (3 percent) were taking some form of neuroleptic medication. Because of their medical problems, many of the people at highest risk have an established relationship with a health care provider; such providers may be able to increase patients' awareness of the dangers of heat and convey messages about preventive measures, even during annual visits that do not occur during hot weather.

Although previous studies have consistently confirmed that there is an excess of deaths due to cardiovascular causes during periods of high or prolonged heat and have supported the biologic plausibility of the exacerbation of existing ischemic heart disease by heat, little information has been available about specific risk factors for heat-related death[3,4,25-31] (and Wainright S, et al.: personal communication). From our study we know that people who died of cardiovascular disease were somewhat older than those whose death certificates listed heat as the underlying or secondary cause of death (average, 76 vs. 70 years; $P<0.001$) and had a greater frequency of preexisting medical conditions (84 percent vs. 73 percent, $P<0.05$). Although the magnitude of risk varied among the cause-of-death categories, we found similar risk factors in these categories. Measures to prevent heat-related death are particularly important for people with cardiovascular disease.

Access to air-conditioned environments is the factor with the greatest protective effect with respect to heat-related mortality.[1,8] We found that people who lived in apartments without air conditioning had a lower risk if they had access to an air-conditioned lobby. These results have important implications for public health. Providing air conditioning to common areas in buildings or in the immediate neighborhood or providing readily accessible transportation to public cooling shelters could help to reduce excess mortality during hot weather.

Excess deaths do not typically occur until the second, third, or fourth day of a heat wave.[7,10] Specific information on how to avoid heat-related illness should be included in media coverage as soon as possible along with meteorologic forecasts of heat waves.

Conclusions

The risk factors identified in this study reflect the complexity of the environmental, social, and medical components of heat-related mortality. Since heat-related deaths are preventable, targeted actions by public health professionals, health care providers, and the media can save lives each summer. People, especially elderly people, who live alone and do not have networks of social contacts and those with debilitating conditions are at particularly high risk during heat emergencies. These people need to be made aware of simple ways they can adapt their daily routines to prevent death due to hot weather; air-conditioned environments should be made readily available and accessible. During the summer heat wave of 1995 in Chicago, anything that facilitated social contact, even membership in a social club or owning a pet, was associated with a decreased risk of death.

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Some Effects of the Urban Structure on Heat Mortality

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During heat waves the death rate from heat-related ailments is often much higher in cities than in outlying environs. The higher death rate in cities appears to be a result of climate modification due to urbanization. Daytime urban–rural climatic differences are small. Nighttime urban–rural climatic differences, however, are often significant—air temperature is warmer within cities at night, long-wave radiant heat load is greater, wind speed is often lower, and inside air temperature of characteristic urban buildings is warmer. Nocturnal urban heat islands of 4–7° are shown to be associated with heat waves in two cities. During heat waves inhabitants of urban areas may experience sustained thermal stresses both day and night while inhabitants of the outlying environs often obtain some relief from thermal stresses during nocturnal hours. It is suggested that excess deaths occurring in urban areas during periods of extreme heat can be significantly reduced through appropriate urban land use.

Man has exhibited great ability to modify his atmospheric environment radically through the process of urbanization. His modification of the environment has often resulted in increased atmospheric stresses being placed upon him. Prolonged exposure to high atmospheric temperatures (e.g., during heat waves) may result in death either as a primary cause or as a contributing factor in heart disease, strokes, and pulmonary disorders (Grover, 1938; Schuman *et al.*, 1964; Bridger and Helfand, 1968). The results of several studies suggest that a large percentage of heat-related deaths may be due to climate modification brought about by urbanization: Shattuck and Hilferty (1932, 1933) found that the urban heat-related death rate is higher than the rural heat-related death rate. The heat-related death rate tends to increase markedly with increase in size of the city. Henschel *et al.* (1968) found that, of the 246 deaths attributed primarily to heat in the St. Louis metropolitan area during July 1966, 85% occurred within the city limits (which contained about 32% of the population); the death rate due to heat within the city limits was 5½ times greater than in the adjacent suburbs. Buechley *et al.* (1972), in a study of the July 4, 1966, heat wave in the New York metropolitan area, found a high correlation between the spatial distributions of excess deaths and of minimum temperature.

The thermal elements of the environment important in maintaining the heat balance of the human body are temperature, humidity, air movement, and radiant energy exchange. All of these thermal elements contribute to mortality

[1] On assignment from the National Oceanic and Atmospheric Administration, U. S. Department of Commerce.

due to heat-related ailments during heat waves (e.g., Root and Stone, 1937). This paper discusses some effects of cities on the thermal elements of the environment.

URBAN CLIMATE MODIFICATION

Among those elements of the climate that exhibit reasonably well-documented urban–rural differences are temperature, solar radiation, cloudiness, humidity, wind speed, visibility, and precipitation. Of direct interest in the study of physiological thermal stresses is that near-surface air temperatures are warmer within cities than in surrounding suburban and rural areas, that air movement is less within cities, that moisture content of the air is slightly lower within cities, and that the solar radiation on a horizontal surface is less within cities. The effects of the urban structure on these four elements of climate are briefly summarized below. Further information is contained in the comprehensive survey of urban climatology by Peterson (1969).

Temperature. Urban climate modification is best exemplified by urban–rural temperature differences. Average long-term annual temperatures for a number of cities of varying sizes are 0.5–1° warmer, and mean winter minima are 1–2° warmer than adjacent rural areas (Landsberg, 1970a). The variations of the urban and rural temperatures are presented for a warm day in Fig. 1 for Dallas, Texas. There is little difference in the maximum temperatures between urban and rural areas. The urban temperature excess reported for day time hours is small and not easily detectable (e.g., see Ludwig and Kealoha, 1968). After sunset the rural surface cools at a more rapid rate than the urban surface, and a relatively large urban–rural temperature difference is normally observed near sunrise. The diurnal pattern of urban–rural temperature differences in Fig. 1 is typical of urban areas; however, the magnitude of the differences will vary as a function at both city size and meteorological conditions.

The horizontal gradients of minimum temperatures in metropolitan areas are often pronounced and give the appearance of the commonly referred to "urban heat island" (e.g., see Fig. 2). The warmest temperatures are normally observed

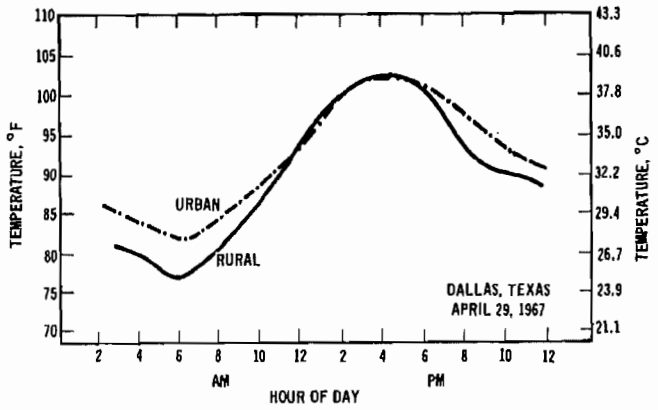

FIG. 1. Diurnal variation of urban and rural temperatures (based on data of Ludwig and Kealoha, 1968).

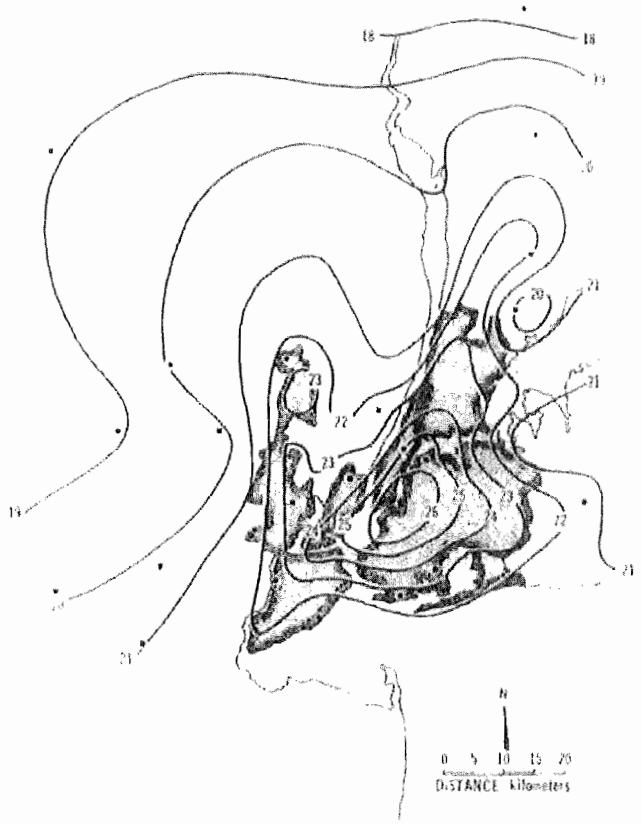

FIG. 2. Analysis of average minimum temperatures (°C) over the New York City metropolitan area July 4, 5, 17, 18, 22, and 23, 1955. Dots indicate location of temperature stations. The shaded areas are cities with population of 50,000 or greater as indicated in the Rand McNally Road Atlas, 45th Edition.

near the center of the city with a strong gradient of temperature in the suburban areas of the city. The magnitude of the urban heat island during the nighttime hours is a function of city size and population density, as indicated in Table I by a comparison of data for three cities (Duckworth and Sandberg, 1954). As cities grow in population and size they also become warmer compared to

TABLE I
COMPARISON OF URBAN HEAT ISLAND AND CITY SIZE[a]

	San Francisco	San Jose	Palo Alto
Population	784,000	101,000	33,000
City area (km²)	116.5	38.6	22.2
Population density (population/km²)	6,730	2,617	1,486
Average urban–rural temperature difference (°C)	6–7	4–5	2–3
Greatest temperature difference (°C)	11.1	8.3	7.0

[a] Based on data by Duckworth and Sandberg (1954).

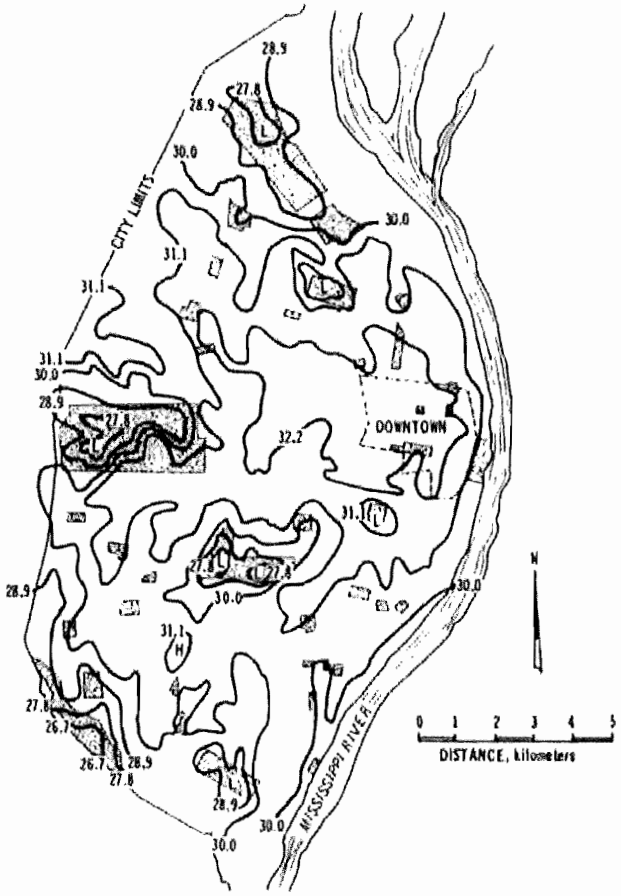

FIG. 3. Temperature pattern (°C) over St. Louis, Missouri, 2200 CST, 9 June 1953. Parks, cemeteries, and other undeveloped lands are shaded (from Stanford University Aerosol Laboratory and the Ralph M. Parsons Company, 1953).

adjacent rural areas. The magnitude of the urban heat island also varies with prevailing meteorological conditions. Clear skies, light surface winds, and low humidity are favorable conditions for a well-developed nocturnal urban heat island.

The urban temperature excess results primarily because of the composition and physical structure of the urban surface. The brick and concrete that make up a high percentage of the urban surface have a higher heat conductivity and storage capacity than the soil and grass that generally comprise the rural surface: the specific heat per unit volume of concrete is about twice that for dry clay or dry sand, and the thermal conductivity of concrete is about 10 times that of dry clay or dry sand. During daylight hours much of the solar radiation striking the rural surface is reflected back into space or is used in evaporative processes or to heat the atmosphere by the processes of conduction and convection from the heated surface. Little of the solar radiation striking the urban surface is used in evaporative processes; because of the high thermal admittance of the urban

surface, much of the solar radiation is effectively stored in the urban structure as heat. After sunset both urban and rural surfaces cool by the radiative process. Because of the large reservoir of heat and counter radiation between buildings, the urban structure cools at a slower rate resulting in an urban temperature excess. Another factor contributing to the urban temperature excess is heat produced within the city by man's activities.

Figure 3 indicates some effects of developed and undeveloped urban land-use areas on the temperature of the overlying air. This detailed analysis of air temperature 2 M above the surface was obtained over St. Louis, Missouri, several hours after sunset on June 9, 1953 (The Stanford University Aerosol Laboratory and the Ralph M. Parsons Co., 1953). The warmest temperatures are over the densely built-up "downtown" area of the city. The large parks and undeveloped areas are almost 5° cooler than the downtown area, and are characterized by a large gradient of temperature as the land-use type changes from urban to undeveloped.

Wind speed. The annual mean wind speed within cities is about 20–30% less than that observed at airport sites in predominately suburban or rural areas (Landsberg, 1970a). The reduction in wind speed within cities results because the variety of sizes of buildings that make up the urban structure effectively retard the movement of air. Observations of wind speed within urban areas are, however, generally made at sites selected to minimize the effect of buildings in the immediate vicinity of the site. The wind speed at street level is probably reduced by a large factor of the airport wind speed. A few measurements of street-level winds (i.e., 2 M above the surface) in comparison to those made at airport sites are presented in Table II. The street-level wind speed is only about 35% of that observed in open fields of nearby airport stations in predominantly suburban or rural settings. However, because of the normally higher elevation of airport wind observations and the extreme roughness of the urban surface, direct comparison of airport–street-level winds are not necessarily indicative of the relative ventilation available to urban and nonurban inhabitants.

Body heat loss by evaporative cooling is directly proportional to the ventilation rate and, consequently, is normally lower in urban areas than in adjacent rural areas. Heat loss through convection from the body is also a function of ventilation rate (and the temperature difference between the skin and the

TABLE II

COMPARISON OF STREET LEVEL WINDS AND AIRPORT WINDS (m/sec)[a]

	Minneapolis		St. Louis		Winnipeg	
	Nocturnal	Daytime	Nocturnal	Daytime	Nocturnal	Daytime
Airport	4.0	3.9	4.0	4.8	4.7	6.1
Street level	1.4	1.1	1.4	1.9	1.3	2.1
% reduction	65	70	66	60	73	65
No. of observations	33	6	26	4	21	11

[a] Based on data presented in: Stanford University Aerosol Laboratory and the Ralph M. Parsons Company (1953).

environment) and is normally less in urban areas, especially during nocturna. hours when air temperature is normally less than skin temperature. Under very light wind conditions the wind speed in urban areas may exceed wind speed in adjacent suburban and rural areas (Clarke and Bach, 1971). The differences are small, however, and the relative effects on the heat-exchange processes are probably not significant.

Moisture content. Evaporative body heat losses are also a function of the moisture content of the air. The moisture content of the air within cities, both relative and absolute, is lower than in adjacent rural areas (Peterson, 1969). The differences in absolute moisture as indicated by wet-bulb temperature and vapor pressure are, however, small (Clarke and Bach, 1971). The slightly lower atmospheric moisture content of urban areas as compared to rural areas results in a minor additional cooling effect on urban inhabitants. The lower moisture content of urban air results from the nature of the urban surface—the streets and sewers afford rapid runoff of precipitation, and the scarcity of vegetation areas provides little opportunity for the addition of moisture through evapotranspiration.

Radiant heat. In many instances, the net radiant heat load (i.e., the total radiant energy, both short- and long-wave, from all sources) is a major element in the body heat load. The main source of radiative heat gain to the body associated with the natural atmospheric environment is solar (short-wave) radiation. Urban areas receive about 15–20% less solar radiation than adjacent rural areas (Landsberg, 1970a). The urban deficit of solar radiation results because of increased cloudiness and atmospheric pollutants in urban areas and is normally greatest during the winter season.

The long-wave radiant heat load is a function of the temperature of the surrounding surfaces (an object radiates heat in proportion to the fourth power of its absolute temperature). Landsberg (1970b) gives infrared thermometer measurements of the surface temperatures of grass, pavement, and buildings in an isolated courtyard. The temperatures of paved areas and brick buildings exposed to the sun during the afternoon hours were reported to be warmer than the air temperature measured in the courtyard by 12° or more. Those buildings and paved surfaces not exposed to the sun were 1.4–5° warmer than the air temperature. In contrast, a grass area exposed to the sun was only 2° warmer than the air temperature. After sunset, the surfaces of the buildings and paved areas were 1–5° warmer than the air temperature, while the grass surface was 2° cooler than the air. Because of the higher density of buildings and paved surfaces in urban areas, the long-wave radiant heat load is typically greater in urban areas than in suburban or rural areas.

Calculations of net radiant heat load during warm summer days are presented by Clarke and Bach (1971) for Cincinnati, Ohio. During afternoon hours, the net radiant heat load directed from the environment to the body was consistently higher above paved surfaces than above grass surfaces. During nighttime hours the net radiant heat load was directed from the body to the environment; however, the heat loss from the body was significantly greater above grass surfaces than above paved surfaces.

Microclimatic effects. The preceding discussion of the urban–rural thermal environments assumes similar outdoor exposure of inhabitants of both areas. Obviously a range of climatic options are available. For example, the radiant heat load will vary significantly from the sunny to the shady side of a building, and ventilation is often higher in urban streets aligned with the prevailing wind. However, climatic options are more readily available to the nonurban inhabitant who, only a short distance from his dwelling, may find the relative comfort of a shaded, grass-covered area exposed to the prevailing wind.

Man spends a large portion of his life inside buildings. The microclimate of characteristic urban and suburban dwellings is another factor, a socioeconomic factor, contributing to the more severe urban environmental heat load during heat waves. The maximum interior daytime air temperature of buildings is generally less than that of the exterior atmospheric environment. During nocturnal hours, however, the thermal energy stored in the building structure is transferred to the immediate environment keeping the interior air temperature of unairconditioned buildings warmer than that of the exterior atmospheric environment (Givoni, 1969). The effect of buildings on their interior temperatures is more pronounced (i.e., lower daytime temperatures and higher nighttime temperatures than that of the exterior atmospheric environment) in massive buildings in urban areas than in single-family suburban housing units. Large buildings in urban areas generally have a higher heat storage capacity than smaller suburban buildings, and because of the normally lower wind speeds in urban areas, less building heat is lost to the atmosphere by convection. Natural ventilation is also less in characteristic urban buildings. Natural ventilation in rooms with only one opening to the environment (common in large urban buildings) is only about 15% of the exterior wind speed. Natural ventilation in rooms with cross-ventilation (common in small suburban dwellings) is about 60% of the exterior wind speed (Givoni, 1969).

The deleterious effects of three of the four thermal elements of the environment (i.e., temperature, ventilation, and radiant heat) are often increased in urban areas; thus, during heat waves, additional and often fatal body stresses may be exerted on urban inhabitants not experienced by those in the adjacent nonurban environs. Of the three thermal elements of the environment acting to increase the effects of heat on urban inhabitants, only measurements of air temperature are normally obtained with sufficient spatial density to allow a quantitative comparison of the urban and rural thermal environments. In the following section heat waves in two cities are discussed with respect to the magnitude of the urban–rural variations of temperatures. This discussion will provide some insight as to the magnitude of the effect of the urban composition and physical structure on heat mortality.

ANALYSIS OF HEAT WAVES

During July 1955, a series of heat waves plagued the New York City metropolitan area. A time series for temperature departure from normal at the Central Park Weather Bureau Station and total daily mortality are presented in Fig. 4 (after Kutschenreuter, 1967). An analysis of the average minimum temperatures

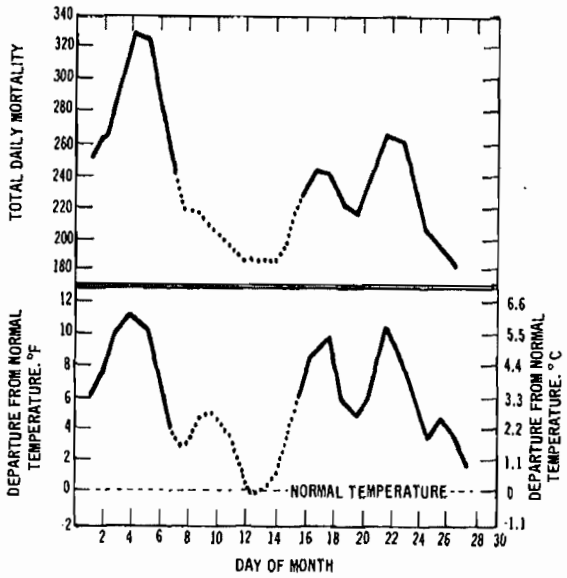

FIG. 4. Three-day running mean departures from normal temperatures (Central Park, New York City) and 3-day running mean values of total mortality for New York City, July 1955. Mortality figures are lagged 1 day behind normal temperature departures (after Kutschenreuter, 1967).

over the New York metropolitan area for July 4, 5, 17, 18, 22, and 23, the days before and the days of the peaks in mortality (mortality data plotted in Fig. 4 is lagged by 1 day), is presented in Fig. 2. The analysis is based on minimum temperature data for 23 stations (indicated by dots in Fig. 2). The highest temperatures are clearly associated with the New York City urban complex. Temperatures over the nonurban areas west and north of the highly built-up inner city are 5–7° cooler. A similar analysis for the maximum temperatures is presented in Fig. 5. However, the warmest maximum temperatures are not associated with the New York City urban complex but are typically displaced inland reflecting the moderating influence of the ocean on air temperature near the shore. The average of the maximum and minimum temperatures are 2–4° warmer over the highly urbanized areas than over the outlying areas.

In the St. Louis metropolitan area between 9 and 15 July 1966, over 500 excess deaths occurred; excessive heat was the primary cause of 246 of the deaths (Henschel *et al.*, 1968). The heat death rate per 100,000 population was 30 in the city but only 5.5 outside the city limits. Average maximum and minimum temperatures for the heat wave are presented in Fig. 6 for nine stations in and around St. Louis. Reasonable maximum or minimum temperature patterns cannot be drawn for the data in Fig. 6 because of the sparsity of stations and because the temperatures at two outlying stations are probably influenced by urban climatic effects. The cities of St. Charles and Alton are sufficiently large to induce their own heat island (see Table I). The temperatures at Weldon Spring and Crystal City are probably indicative of the rural environs surrounding St. Louis; the minimum temperatures in the rural environs are 4–5° cooler than

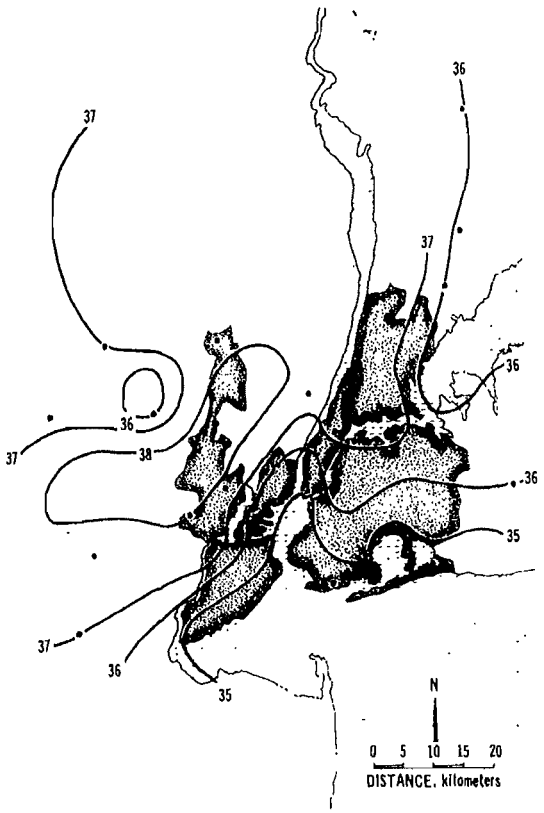

FIG. 5. Analysis of average maximum temperatures (°C) over the New York metropolitan area July 4, 5, 17, 18, 22, and 23, 1955. Dots indicate location of temperature stations. The shaded areas are cities with population of 50,000 or greater as indicated in the Rand McNally Road Atlas, 45th Edition.

in the city. There is no apparent difference in the maximum temperatures of the rural and urban environs.

DISCUSSION

Heat-related deaths usually do not occur until after exposure to heat wave temperatures for 24 hours or more (Henschel *et al.*, 1968; Kutschenreuter, 1967; Shattuck and Hilferty, 1933). This and the analysis presented herein suggest that higher thermal stresses within cities, particularly the lack of adequate relief from thermal stresses at night, may be a significant factor causing the urban mortality rate during heat waves to be much higher than the suburban and rural mortality rates. Adequate relief from thermal stresses is often naturally available during the nocturnal hours to those living in rural areas while urban inhabitants may experience thermal stresses day and night.

Nonthermal factors undoubtedly contribute to the apparent higher death rate in urban areas during heat waves. The generally more affluent inhabitants of suburban communities are more likely to have, in addition to better housing, a higher percentage of airconditioned housing than inhabitants of urban areas.

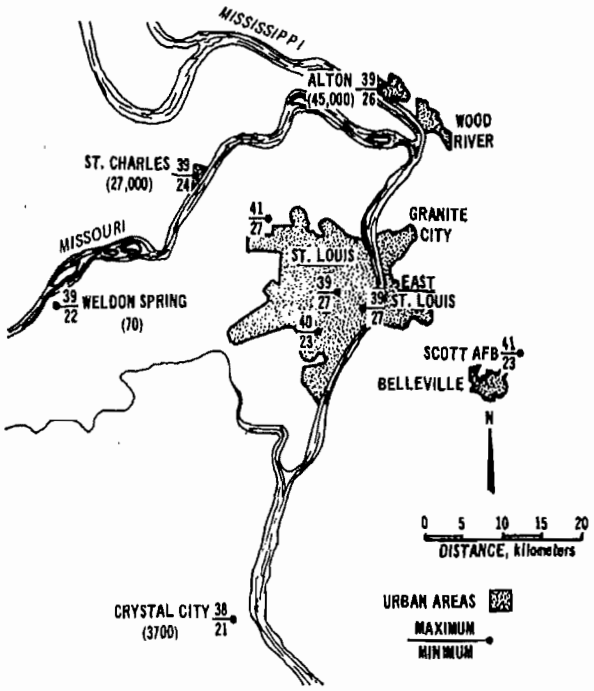

FIG. 6. Average maximum and minimum temperatures (°C) 9–15 July 1966, in the St. Louis metropolitan area (population in parentheses for outlying cities with temperature data).

Education, i.e., knowledge of health precautions appropriate to hot weather, availability of medical care, extent of physical activity, type of clothing, etc., often differs in urban and nonurban areas and may contribute to the higher urban heat-related death rate.

Air pollution could also be a factor in heat-related deaths. During the heat waves reported in this paper, average wind speeds were slightly below the long-term average July wind speeds, and both cities received more than 85% of possible sunshine. Oxidant concentrations were probably above normal although it is unlikely that they were sufficiently high as to directly affect mortality. Air pollution is, however, an environmental stress, which, in combination with extreme heat stress, may contribute to the increase in mortality. The distribution of pollution over a city is similar to the distribution of nocturnal temperatures (i.e., the highest concentrations are normally found over the center of the city with relatively lower concentrations in the suburban areas). Consequently any contribution of air pollution to the elevated death rate during heat waves is masked by the dominant thermal effects and difficult to demonstrate.

Urbanized land area is only about 1% of the total land area of the United States. However, almost 65% of the population of the United States live in urbanized areas and experience to some degree the effects of urbanization on climate. Over 29% of the population live in central cities and may be exposed to the urban thermal anomaly as discussed herein. Aside from mortality effects are the esthetic and sociological consequences of the urban thermal anomaly which contributes to the general inhospitable summer climate of many large cities and

the effects of heat on mental attitude, performance, and behavior as noted by Landsberg (1969).

The urban thermal anomaly can be expected to increase if man continues to expand his cities into giant metropolitan regions. Because of the increasing use of airconditioning, the portion of the population of cities necessarily exposed to thermal stresses will decrease. The heat and moisture removed from the buildings, however, will be dumped into the urban atmosphere and further increase the thermal anomaly of the city. This will also increase thermal stresses on the inhabitants of the central city not fortunate enough to have airconditioning. This increase in contrast of personal exposures could increase the heat-related death rate in the lower economic classes. Air and thermal pollution associated with the energy requirements for airconditioning will also increase although they will not necessarily directly affect urban inhabitants.

The urban thermal environment can be partially controlled through appropriate urban land use. The adequate provision of green areas judiciously spaced over the metropolitan area is one example. The effect of green areas on the nocturnal thermal climate of cities is substantial (see Fig. 3). The heat surplus in cities may also be diminished by the use of building materials of low heat conductivity and storage capacity, and of high albedo. Siting of urban activities with respect to micro- and mesoclimates is another avenue open to the planner; the heat stress on occupants of housing units without airconditioning would be considerably less in suburban areas or on the fringe of a large park than if they are located in the heart of the urban complex.

SUMMARY

The atmospheric environment over cities is modified from that over adjacent nonurban environs. During the summer, urban climate modification often results in additional thermal stresses on inhabitants of urban areas not experienced by inhabitants of adjacent nonurban environs. During heat waves, the death rate due to heat-related ailments is often significantly higher in urban areas than nonurban areas.

Daytime urban–rural climatic differences are small; the air temperature of urban areas is increased slightly, wind speed is decreased, humidity is slightly lower, the radiant heat load is greater above paved surfaces than above grass, and the inside air temperture of characteristic urban buildings is lower. Nighttime urban–rural climatic differences are often significant; air temperature is warmer within cities, the long-wave radiant heat load is greater, wind speed, except on near-calm nights, is lower, humidity is slightly lower, and the inside air temperature of urban buildings is higher. Thermal stresses are often significantly greater in urban areas than nonurban areas because of the net effect of these factors.

The spatial variation of near-surface air temperatures was examined for heat waves that resulted in a large number of excess deaths in two metropolitan areas. There was little apparent difference in the maximum daytime temperatures of the urban and rural environments. At night, however, a well-developed heat island was obvious for both metropolitan areas; air temperatures over the densely

built-up urban areas were 4–7° warmer than air temperatures over the outlying suburban and rural areas. The additional environmental thermal stresses on urban inhabitants will increase as cities become larger. However, they can be controlled to a degree through appropriate urban land use.

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The Potential Impacts of Climate Variability and Change on Temperature-Related Morbidity and Mortality in the United States. By: McGeehin, Michael A.; Mirabelli, Maria. Environmental Health Perspectives Supplements. May2001 Supplement 2, Vol. 109, p185. 5p. 1 Diagram, 1 Graph. Abstract: Heat and heat waves are projected to increase in severity and frequency with increasing global mean temperatures. Studies in urban areas show an association between increases in mortality and increases in heat, measured by maximum or minimum temperature, heat index, and sometimes, other weather conditions. Health effects associated with exposure to extreme and prolonged heat appear to be related to environmental temperatures above those In which the population is accustomed. Models of weather-mortality relationships indicate that populations in northeastern and midwestern U.S. cities are likely to experience the greatest number of illnesses and deaths in response to changes in summer temperature Physiologic and behavioral adaptations may reduce morbidity and mortality Within heat-sensitive regions, urban populations are the most vulnerable to adverse heat-related health outcomes The elderly; young children, the poor, and people who are bedridden or are on certain medications are at particular risk Heat-related illnesses and deaths are largely preventable through behavioral adaptations, including the use of air conditioning and increased fluid intake. Overall death rates are higher in winter than In summer, and it is possible that milder winters could reduce deaths in winter months However, the relationship between winter weather and morality rs difficult to interpret. Other adaptation measures Include heat emergency plans, warning systems, and illness management plans Research is needed to identify critical weather parameters, the associations between heat and nonfatal illnesses, the evaluation of implemented heat response plans, and the effectiveness of urban design in reducing heat retention Key words: climate change, cold weather, global warming, heat waves — Environ Health Perspect 109(suppl 2):185–189 (2001). http://ehpnet1.niehs.nlh.gov/docs/2001/suppl-2/185-189mcgeehin/abstract.html [ABSTRACT FROM AUTHOR] (*AN: 5142370*)

Database: Academic Search Complete

The Potential Impacts of Climate Variability and Change on

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The relationship between human health and stressful weather is a complex medical, social, and environmental issue (Figure 1). Future-climate scenarios suggest that higher global mean temperatures could result in marked changes in the frequency of temperature extremes (1). Software developed by the National Oceanic and Atmospheric Administration (2) can be used to estimate changes in the probability that a given extreme temperature will occur for a specified duration under a defined climate scenario.

Heat-Related Health Risks

Heat waves are sporadic but recurrent. Elevated temperatures during summer months are associated with excess morbidity and mortality. Conservative estimates are that, on average. 240 hear-related deaths occur annually in the United States; in a 1980 heat wave, there were 1,700 deaths (3). Following a 5-day heat wave in 1995 in which maximum temperatures in Chicago, Illinois, ranged front 93 to 104°F, thc number of deaths reported increased by 85%, and the number of hospital admissions increased by 11% compared with numbers recorded during the same period in the preceding year (4–6). During this period at least 700 excess deaths (i.e., deaths beyond those expected (hr that period in that population) in Chicago were recorded, most of which were directly attributed to heat (4–6).

Exposure to extreme and prolonged heat is associated with heat cramps, heat syncope (fainting), heat exhaustion, and heatstroke (7). The initial human physiologic response to heat entails increasing surface blood circulation, thereby promoting heat loss through radiation, convection, perspiration, and increased

rates of evaporative cooling (8). The ability to respond to heat stress is limited by the capacity to increase the maximum cardiac output required for cutaneous blood flow. The cardiac output, in turn. is a function of maximal heart rate, intravascular volume, and sustainable renal and splanchnic vasoconstriction. Under mild heat stress, heat acclimatization can increase the body's tolerance to heat stress. However, under extreme or chronic heat stress, the body loses its ability to maintain temperature balance and death may occur.

The most common cause of death and the most acute illness directly attributable to heat is heatstroke, a condition characterized by a body temperature of 105.0°F (40.6°C) or higher and altered mental status (9). Other causes of death observed to increase following hear waves include ischemic heart disease, diabetes, stroke, respiratory diseases, accidents, violence, suicide, and homicide (10, 11). Even when appropriate, medical examiners do not routinely record these causes of death as heat related.

As observed following a 1980 beat wave in Kansas City, Missouri, heatstroke victims showed few symptoms of illness prior to the onset of bear stroke (12). The onset of heatstroke occurs rapidly through progressively serious symptoms, including lethargy, confusion, disorientation, delirium, and coma (4, 13). Survivors of heatstroke often experience persisting organ dysfunction that is pre dietive of 1-year mortality (i.e., death within 1 year of an event) (14). Hear stroke mortality and heat-related mortality from all causes appear to peak with a 1- to 2-day lag folling high temperatures (6.15.16). One epidemiologic study of deaths during and following a heat wave indicated that a rise in the hear index (HI) is followed by an increase in the number ef deaths due m heat (Figure 2) (6),

The impact of heat on morbidity is less certain than the heat-mortality association (17.18). A 5% increase in hospital admissions was observed during the 1980 heat wave in Kansas City (12). During periods of excessive heat. emergency rooms report an overall increase in visits, specifically for fainting, nausea, dizziness, and heat cramps (16, 19). A detailed analysis of all inpatient hospital admissions during the 1995 heat wave in Chicago found that individuals with a wide range of underlying medical conditions were at increased risk for hospitalization. These underlying medical conditions included cardiovascular and respiratory diseases, diabetes, renal diseases, nervous system disorders, emphysema, and epilepsy (5, 20). Increases in hospital visits for cardiovascular diseases (11, 16) and increases in deaths due to cardiovascular and respiratory diseases (2l) also have been documented during heat waves, suggesting that heat exacerbates these conditions. Dehydration and volume depletion limit the cardiovascular response necessary to increase the cutaneous circulation during heat stress. Consequently, patients with underlying diseases or the elderly may not have the physiologic capability to adequately respond to heat exposure.

Meteorologic Conditions and Associated Health Conditions

A scientific question currently being investigated pertains to the meteorologic air masses present during heat waves and how they can predict adverse effects. In a recent study Kalkstein and Greene (22) used a new air mass-based synoptic procedure to evaluate his historical weather-mortality relationships and to

illustrate how climate change scenarios might alter these relationships. Data from 44 large U.S. cities were analyzed for air masses identified in each city, and for each air mass the weather-mortality relationship was estimated. Climate change scenarios were then applied to each weather-mortality relationship to estimate the potential changes in the relationship by 2020 and 2050. Using three unique models and accounting for possible acclimatization, Kalkstein and Greene concluded that, under the proposed climate scenarios, summer mortality will increase markedly, whereas winter mortality will decrease slightly.

Rates of morbidity and mortality due to heat-related illnesses can rise during and immediately after a heat wave. However, thc magnitude of these health effects is difficult to predict and depends on a variety of factors such as suddenness of heat onset, city planning for heat emergencies, regional heat tolerance, heat duration, and macroclimate adaptability. The sudden onset and magnitude ora heat wave early in summer in a city with no prior experience with extreme weather conditions, and thus no detailed beat emergency plans, can pose serious health risks.

In the United States, there does not appear to be a single universal threshold temperature above which rates of heat-related morbidity and mortality rise sharply. Instead. tolerance of excess heat varies regionally according to the population and its preparedness for heat and according to the local average temperatures and frequency of extreme temperatures (23). In temperate regions, severe but infrequent temperature fluctuations, such as very hot episodes during periods of generally milder weather conditions, are associated with increases in weather-related mortality. In tropical regions, summer temperatures are higher for a longer period of time and are less variable. As a result, elevated temperatures in these regions do not appear to have a significant impact on weather-related mortality.

Models of the weather-mortality relationship indicate that populations in northeastern and midwestern U.S. cities may experience the greatest number of heat-related illnesses and deaths in response to changes in summer temperature, and that the must sensitive regions are those where extremely high temperatures occur infrequently or irregularly (24). including Philadelphia, Pennsylvania (25'); New York, York, New York (ll); Chicago, Illinois (4); Milwaukee. Wisconsin (13); and St. Louis, Missouri (17), where past heat waves resulted in large numbers of heat-related deaths. In some heat waves, daily mortally levels are more than double baseline levels (26). The most severe conditions, resulting in the highest heat-related mortality rates, are often characterized by successive days of high temperatures coupled with high overnight temperatures (27). Furthermore, the duration of the heat. including overnight heat, and the microclimate of non-air-conditioned automobiles, apartments, and other dwellings appear particularly critical to the heat mortality relationship (18).

Risk Factors for Heat-Related Illnesses

Major risk factors for heat-related morbidity and mortality include urban living, age, and socioeconomic factors, as well as preventive behaviors.

Urban Living

Urban living is an important risk factor. Populations residing on the top floor of apartment buildings (6.28), in urban areas (12), and without access to air-conditioned environments (6.28) experience higher rates of heat-related morbidity and mortality, suggesting that the living conditions of susceptible populations are important.

The "urban heat island effect" describes the elevated temperatures observed in urban areas. Observational studies indicate that the HI and heat-related mortality rates are higher in the urban core than in surrounding areas (29). Buechley et al. (30) attributed the elevation in heat-related deaths in urban areas to the high population density. Clarke suggested that urban areas retain heat throughout the nighttime more efficiently than the outlying suburban and rural areas (31). The overnight heat may create a critical thermal stress (i,e., no nighttime relief from heat) to urban inhabitants, resulting in excessive hear-related deaths.

The importance of living environments in the weather-health relationship is supported by recent analyses (32, 33). The findings of an analysis of hot weather-related morality across tile United States indicate that household conditions in urban and metropolitan areas, including central air conditioning and other indicators of housing quality, may have an important impact on heat-related health outcomes (32). During a heat wave in St. Louis, higher mortality rates were retarded predominantly in the business and urban core areas than in other, cooler sections of the city (3.3). According to the U.S. Bureau of the Census, 29% of the occupied housing units in the United States in 1995 were located in the central cities, 35% in the suburbs. 20% in nonmetropolitan areas, and 13% in rural areas (34.35). Jones et al. (12) examined mortality following a July 1980 heat wave and found that deaths from all causes increased by 57 and 64% in the major metropolitan areas of Sc Louis and Kansas City, respectively, but only by 1(1% in predominantly rural areas in the remainder of Missouri.

Age

Another risk factor is age. Epidemiologic studies of heat-related morbidity and mortality during and following heat waves suggest that the elderly and young children are particularly vulnerable (9, 12, 36, 37). Regardless of race or gender, individuals 65 years of age or older are more susceptible to the adverse effects of heat than are younger adults (5, 9, 27, 38). The risk for heat related death increases sharply with age, as those 85 years of' age or older are most at risk for heat-related mortality (9). Like the elderly, young children are at high risk for heat-related illnesses (9). Young children with certain predisposing illnesses such as diarrhea, respiratory tract infections, and neurologic defects are at especially increased risk for hyperthermia during extreme heat (12). Both the very old and the very young tend to have reduced heat-regulating mechanisms (9). In addition, each of these populations experiences restricted mobility, resulting in diminished control of their environments, including access to fluids (9).

Socioeconomic Factors, Ethnicity,% and Race

Other risk factors include poverty, social isolation, inadequate English language skills, residence in high-crime areas, certain medications associated with aging, and lack of access to media (such as television and newspapers), and thus reduced awareness both of the potential dangers from heat exposure and of the ways to reduce risk. Populations uniquely vulnerable to the impacts of excessive heat exposure include the poor (16, 39) and the socially isolated (6). Populations of lower socioeconomic status may not have access to air-conditioned places because of thc cost of an air-conditioning unit or utility hills (6, 16). Although opening windows and using Fans for ventilation may help reduce stagnant and hot indoor air conditions, the effectiveness of electric fans in reducing tile risk of heat-related mortality has not been substantiated (6). Individuals confined to bed or unable to care for themselves are at increased risk (6, 28). whereas those living with others are at decreased risk and are more likely to increase their fluid intake during heat waves. Non English-speaking or -reading populations, who exemplify the group of individuals lacking access to heat-relieving conditions, are quire vulnerable. Furthermore, because of neighborhood crime or violence, urban residents, including the elderly, report a reluctance to leave windows open (16, 40). Finally, patients taking medications or drugs that modify thermoregulatory capacity are at increased risk also (28, 41, 42). The urban elderly experience the highest rates of heat-related morbidity and mortality because of both their declining thermoregulatory abilities and the extreme heat conditions in urban areas (16).

Data on the 1995 heat wave in Chicago indicate that mortality among African Americans was 50% higher than among whites (38). The disparity likely reflects residence in inner-city neighborhoods, poverty, housing conditions, and medical conditions (12, 16.28). Similar findings emerged following heat waves in Texas (36), Memphis, Tennessee (16). St. Louis. and Kans,ts City (12).

Current Protections Air Conditioning

Air conditioning is often recommended and used to mitigate many of the factors that increase the risk of heat-related illness and death (3.4.13). Over 70% of residences in the United States are now equipped with air conditioning: 46% are equipped with central air conditioning, and 27% are equipped with room air-conditioning units. The proportion of housing units with central and/or room air conditioning varies regionally and ranges from 27% in the Northeast to 88% in the South. In central cities the proportion of residences with air conditioning is nearly identical to that of residences in the entire United States; 71% reported air conditioning equipment (34.35). The use of air-conditioning systems in homes, workplaces, and vehicles has increased steadily over the past 30 years and is projected to become nearly universally available in tile United States by 2050 (34.35).

Behavioral Changes

Peer-reviewed literature contains extensive evidence that heat-related illnesses are largely preventable through behavioral adaptations, including the use of air conditioning (3) increased fluid intake (28) in high-risk populations, although thc magnitude of mortality reduction cannot be predicted.

Community-Wide Planning and Warning Systems

For decades, investigators have asserted the benefits of community-wide heat emergency plans (4, 14, 43,.44), improved heat warning systems (ll,45), and better management plans for heat-related illnesses (11, 46), The ability to adapt to chronic heat stress may hinge upon development of these systems, rather than on changes in thermoregulatory functions. Adaptation and, ultimately, prevention of increases in heat-related morbidity and mortality may be determined by thc ability to caution and educate populations and to prepare for heat emergencies, taking into account risk factors (e.g., medications, age, or residence location) and populations at risk (e.g.. elderly, young, or chronically ill).

Future Adaptations Heat Emergency Response Plans

Chicago. St. Louis. and several other U.S. cities maintain comprehensive beat emergency response plans involving key components: a) preparations prior to the onset of excessive heat, b) meteorology-based warning systems, d rapid and coordinated actions during the heat wave, d) criteria and procedures for deactivating the plan, and d evaluation following thc response activities and outcomes. Preparations include strategies to encourage organizations participating in the heat response activities to review their emergency plans, update a database of at-risk individuals and populations, ensure the availability of resources, and activate record-keeping procedures at all affected agencies. Following the 1995 Chicago hear wave, existing state and community intervention strategies were identified. Assessment of these strategies resulted in the recommendation that a model response plan involving multiple and diverse agencies be developed and disseminated (47). A comprehensive response plan should use information on risk factors with meteorologic variables, prevention strategies, and outcome measurements to monitor and attenuate the impact of excessive heat, This proactive approach to reducing heat-related morbidity and mortality appears to be an effective government-sponsored adaptation to extreme heat that might serve as a model For other communities.

Use of Risk Factors

Extensive epidemiologic research into the risk factors associated with heat-related mortality demonstrates that the relationship between health and temperature is multifactorial. Future use of risk factors to identify the most vulnerable populations is an important strategy to avoid heat-related mortality.

Environmental and Behavioral Measures

Because some degree of projected elevation in worldwide temperatures over the next several decades cannot be prevented, environmental and behavioral changes may be the most effective means of reducing the severe impact of heat. Monitoring the hydration status of the elderly, very young, and medically at-risk populations may indicate when these populations are in danger of dehydration and other, more severe, heat-related illnesses, Organized programs to check on the elderly are frequently arranged by community, volunteer, and religious organizations. Visiting the elderly who live alone or who are socially isolated ensures that they are aware of the dangers of extreme heat and are raking sufficient precautionary measures. Opening air-conditioned shopping malls, community centers and recreation Facilities, and extending the hours of public swimming pools increase thc availability of cool environments. Providing transportation to die facilities may be necessary to ensure that the isolated and

at-risk populations use these environments. These and other measures, such as limiting strenuous activities during peak daytime hours and increasing fluid intake, exemplify environmental and behavioral measures likely to be effective in reducing the impact of hear in high-risk populations (5, 6).

Cold Weather Mortality

An increase in mean global temperatures could result not only in warmer average summer temperatures but also in slightly warmer average winter temperatures. Overall mortality is generally higher in winter than in summer, but there is little convincing evidence that weather patterns are solely responsible. The adverse health effects of cold weather include direct effects, such as hypothermia, and indirect effects, such as increased rates of pneumonia, influenza, and other respiratory illnesses (22). Cold weather-related deaths directly attributable to cold exposure are prevalent in northern and mountainous regions of the United States, but they also occur in the milder climates of the Southern States (48). Hypothermia deaths are reported to have occurred on days with minimum temperatures above 32°F (O°C). Often, alcohol and drug use contribute to hypothermia deaths (48). The impact of an increase in global mean temperature on cold temperature-related morbidity and mortality has not been quantified; however, because hypothermia deaths occur even in Southern States and at temperatures above freezing, a significant reduction in mortality From the direct effects of cold seems unlikely.

Colder regions of the United States experience more frequent cold spells, lower temperatures, and higher levels of snowfall. The lower temperature and/or higher snowfall is often followed by an increased number of deaths due to ischemic heart disease, cerebrovascular disease, and respiratory disease (49). These indirect effects of cold temperature appear to be more severe when accompanied by strong winds (50).

An increase in average winter temperatures could result in a reduction in deaths during winter months, particularly in areas with relatively colder climates. However, many winter-time deaths are due to infectious diseases such as influenza and pneumonia (51). If increases in worldwide mean temperatures encourage people to spend more time outdoors or to increase indoor ventilation, infectious disease transmission may be reduced. European studies indicate an association between mortality and cold temperature, controlling for influenza. This finding suggests that influenza contributes to, but does not fully explain, the association between winter temperature and mortality (50, 52).

An analysis of weather and mortality data in 44 U.S. cities found a weak association between winter weather and mortality (22). The authors used models to estimate climate change for 2020 and 2050 and concluded that winter mortality will decrease slightly but will not offset the larger projected increases in summer mortality. Additional research is needed to understand the relationship between temperature and the predominant causes of death in winter.

Research Needs and Data Gaps

More information is needed about which weather parameters are important in the relationship between

weather and health. Maximum temperature, minimum temperature, relative humidity, HI, and duration of exposure are currently used to estimate exposure to heat. Further research into determining the importance of each of these factors will improve estimation of the relationship between heat and health and facilitate precautionary measures as the thresholds of the key parameters are reached.

More information is also needed about the association between heat and morbidity. Most susceptible are patients with certain chronic medical conditions, such as cardiovascular and cerebrovascular diseases, diabetes, respiratory and renal diseases, Parkinson's disease, Alzheimer's disease, and epilepsy (5, 20). These conditions predispose patients to dehydration, heat exhaustion and heat stroke. Increased awareness among healthcare providers that individuals with these conditions are at higher risk allows for guided intervention. The effectiveness of such tailored prevention efforts should be closely monitored. Surveillance of morbidity during the onset of extreme heat may assist in identifying other susceptible populations. Rydman et al. (19) proposed using computerized data from emergency departments to analyze hospital admissions in real time. However, whether this information can be used to predict heat-related morbidity and mortality has been questioned (53).

Individuals who lack social networks pose a particular challenge for prevention efforts (6). The feasibility and effectiveness of specialized health education efforts aimed at reaching these populations should be evaluated.

To improve the comparability of data collected during and following periods of extreme heat, methods of recording heat-related health outcomes should be standardized. After the 1995 heat wave in Chicago, the Centers for Disease Control and Prevention (3) recommended the use of uniform criteria for the diagnosis of heat-related deaths. Since that recommendation, Donoghue et al. (54) published diagnostic guidelines and criteria for use by medical examiners.

Extensive epidemiologic research of risk factors for heat-related morbidity and mortality offers numerous possibilities For the development of effective interventions. In many cities and regions of the United States, interventions have been established to attenuate health impacts of heat; few, if any, of these interventions have been used during a heat wave or evaluated following their use. Evaluation of existing and implemented heat response plans could determine the cost-effectiveness and health impact of these plans, as well as highlight areas where improvements are necessary.

To assist the efforts of municipalities in their heat wave response plans, we need a greater understanding of the importance of urban design to heat. Although some types of buildings (e.g., brownstones, tail apartment buildings) retain heat efficiently, other urban characteristics (e.g., tree cover and light-colored roofs) may facilitate wind, shade, and other heat-relieving conditions. Research and subsequent incorporation of these infrastructure characteristics into urban areas may contribute to a reduction of the urban hear island effect and its associated health effects.

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DIAGRAM: Figure 1. Protecting the influence of weather and climate change on temperature-related illnesses

GRAPH: Figure 2. Relationship of HI to heat-related deaths, showing probable cause and effect and a lag time of approximately 2 days, Chicago, 11 23 July 1995 Data from the National Oceanic and Atmospheric Administration (47).

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Outdoor Air Temperature and Mortality in the Netherlands: A Time-Series Analysis

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Death rates become progressively higher when outdoor air temperature rises above or falls below 20–25°C. This study addresses the question of whether this relation is largely attributable to the direct effects of exposure to heat and cold on the human body in general, and on the circulatory system in particular. The association between daily mortality and daily temperatures in the Netherlands in the period 1979–1987 was examined by controlling for influenza incidence, air pollution, and "season"; distinguishing lag periods; examining effect modification by wind speed and relative humidity; and distinguishing causes of death. Important direct effects of exposure to cold and heat on mortality were suggested by the following findings: 1) control for influenza incidence reduced cold-related mortality by only 34% and reduced heat-related mortality by 23% (the role of air pollution and "season" was negligible); 2) 62% of the "unexplained" cold-related mortality, and all heat-related mortality, occurred within 1 week; and 3) effect modification by wind speed was in the expected direction. The finding that 57% of "unexplained" cold-related mortality and 26% of the "unexplained" heat-related mortality was attributable to cardiovascular diseases suggests that direct effects are only in part the result of increased stress on the circulatory system. For heat-related mortality, direct effects on the respiratory system are probably more important. For cold-related mortality, the analysis yielded evidence of an important indirect effect involving increased incidence of influenza and other respiratory infections. *Am J Epidemiol* 1993;137:331–41.

air pollution; cause of death; influenza; mortality; seasons; temperature; weather

Several studies have observed a V-like relation between mortality and outdoor air temperature: Death rates are lowest on days with maximum temperatures of approximately 20–25°C and become progressively higher when weather gets hotter or colder (1–6). This pattern suggests that exposure of the human body to unfavorable ambient air temperatures is not just uncomfortable but creates a direct threat to human survival.

The idea that exposure to unfavorable temperatures leads to death directly (i.e., without mediation of other environmental factors) has been supported by the results of thermophysiologic experiments. Particular importance has been given to cold- and heat-induced stress on the circulatory system. In laboratory situations, exposure to high temperatures was found to increase blood viscosity and blood cholesterol levels (7, 8). Exposure to cold results in increases in blood pressure, blood viscosity, and heart rate (8–11). Increases in blood pressure, fibrinogen concentrations, and blood viscosity during the winter season suggest that cold-induced

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cardiovascular stress is quite prevalent in the general population (12, 13).

It is uncertain, however, to what extent excess mortality on hot and on cold days is attributable to exposure to low or high temperatures as such. Other environmental factors might play an equally or more important role. It has been argued that mortality is associated with cold weather because of increased incidence of influenza and other respiratory infections (4, 14–17), increased levels of winter smog (18), or "season" (14, 19–22). The latter variable is usually understood to be a conglomerate of factors, with seasonal variation, such as diet, physical exercise, psychosocial stress, photoperiod, and general body strength. Mortality increases on hot days might result in part from increased photochemical smog (23).

The purpose of our study is to obtain indications on the mechanisms that underlie the association between outdoor air temperature and mortality by means of a detailed analysis of this relation in the Netherlands from 1979 through 1987. The question that we addressed is whether this relation is largely attributable to direct effects on the human body of exposure to unfavorable temperatures. Indications of direct effects will be obtained in three ways:

1. Distinction of lag periods. Since there is no long lag time between exposure to unfavorable outside air temperatures and response of the human body to cold or heat, direct effects might be expected to begin to evolve rapidly. The interval between exposure to unfavorable outside air temperature and death, therefore, depends on the time needed for a fatal outcome to develop once a pathologic process has been induced. This may take more than a week, but it may be expected that a large portion of the deaths will occur more rapidly. Therefore, if most of the effect of exposure to cold and to hot weather on mortality occurs rapidly, this would be suggestive of direct effects of exposure to unfavorable temperatures.

2. Control for influenza incidence, air pollution level, and season. As outlined above, these factors constitute some alternative explanations of the relation between temperature and mortality. However, if they explain only a small part of this relation, this would increase the likelihood that heat- and cold-related mortality is largely attributable to the direct effects of exposure to unfavorable temperatures.

3. Examination of effect modification by wind speed and relative humidity. Thermophysiologic laws state that the effect of exposure to cold on the heat balance of the human body is increased by strong air flow. The effect of heat is increased by weak air flow (which reduces body cooling) and high relative humidity (which reduces evaporation of sweat and, thus, impairs body cooling) (8, 9, 24–26). Therefore, it would be consistent with the idea that the direct effects on the human body play an important role if it were found that the effect of cold weather is greater when cold is accompanied by strong wind and that the effect of hot weather is greater when heat is accompanied by high relative humidity and weak wind.

If direct effects of cold and heat on the human body play an important role, a subsequent question is to what extent such effects result in stress on the circulatory system. This will be examined by distinction of causes of death. It is reasonable to expect that cold- and heat-induced cardiovascular stress increases the death rates of patients with cardiovascular disease more than it does among other patients.

This presentation will not focus on specific heat waves or spells of severe cold, but will consider all days in the study period. Because temperatures in the Netherlands are usually moderate (the Dutch climate is maritime), the results cannot be applied without reserve to periods of extreme heat or cold nor to regions with more extreme climates. This study is primarily intended to shed light on the large number of deaths that are associated with moderate heat and cold in industrialized countries.

MATERIALS AND METHODS

Materials

The Netherlands Central Bureau of Statistics supplied data on the number of deaths

TABLE 1. Causes of death selected for analysis, the Netherlands, 1979–1987

Cause of death	ICD-9* code	Daily no. of deaths		
		Average	Standard deviation	
			Observed	Due to random variation†
All causes	All	324.72	32.86	18.02
Neoplasms	140–208	88.67	10.98	9.42
Cardiovascular diseases	380–459	143.54	19.32	11.98
Respiratory diseases	460–519	16.31	6.25	4.04
All other diseases	Rest, 001–799	60.05	11.07	7.75
External causes	800–998	16.15	4.41	4.02

* ICD-9, *International Classification of Diseases*, Ninth Revision.
† Calculated as the square root of the average number of deaths, assuming that numbers of deaths follow the Poisson distribution.

according to cause and date, from January 1, 1979, to December 31, 1987. Table 1 provides basic information on selected causes. For all causes, the observed daily mortality variation exceeds that which could be expected on the basis of random fluctuations alone.

Age- and sex-specific numbers of person-days at risk for January 1 of each year of the study were obtained from the continuous population registry. Person-days at risk for other days of the year were estimated by means of linear interpolation.

The Netherlands Royal Meteorological Institute supplied data on 24-hour average temperature, wind speed, and relative humidity. All data were referenced to the De Bilt station, which is centrally located. Climatic differences within the Netherlands are small and similar weather changes usually occur on the same day in all parts of the country.

Data on the incidence of influenza and influenza-like conditions were collected from a monitoring network of 45 general practices located throughout the country (27). Cases without virologic confirmation were also counted. Data from England and the United States showed that variation in the incidence of influenza-like conditions accurately reflects epidemic upheavals in the incidence of influenza sensu strictu (28, 29). We found, in an analysis reported elsewhere, that during influenza epidemics, mortality peaks in the Netherlands can be predicted accurately from the incidence of influenza-like conditions (30).

Air pollution was measured by the amount of sulphur dioxide in the air because ample validated data on sulphur dioxide was available for the entire study period. These data were available because a network of measuring stations continuously monitor the concentration of pollutants in the air (31). The daily exposure of the Dutch population as a whole was estimated by using the arithmetic average of the 24-hour average values of six stations which together provide good coverage of the entire country. We reported elsewhere on the association between air pollution and mortality (32).

Methods

By Poisson regression analyses, the daily number of deaths was related to temperature and other independent variables controlling for long-term mortality trends and changes in the age and sex structure of the population.

Each variable of the regression model was checked for a nonlinear relation to mortality by inspection of scatterplots and the addition of a square term to the regression model. A clearly nonlinear relation was found for temperature only. Figure 1 shows a V-like relation, with the lowest mortality occurring when the average daily temperature is about 16.5°C, and approximately linear relations on either side of that point (the

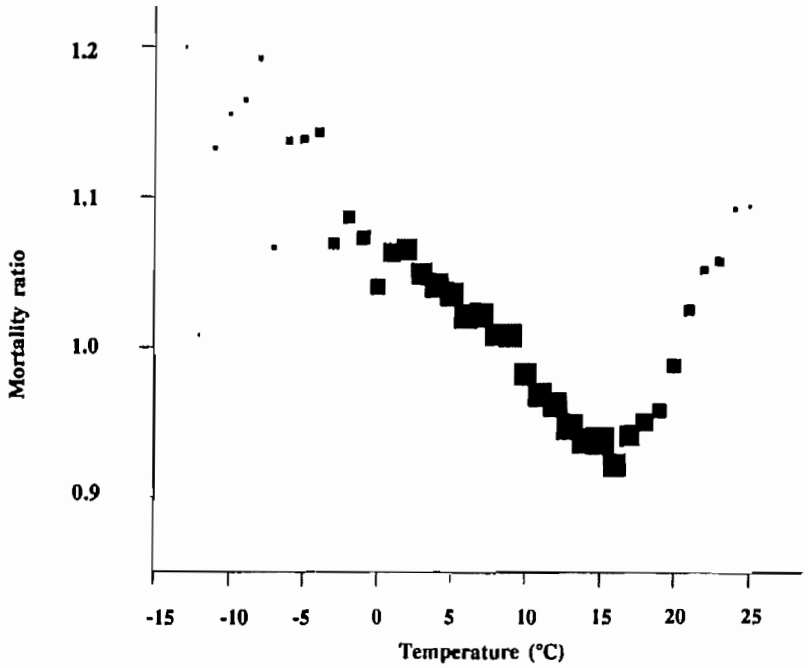

FIGURE 1. The mean mortality level at days grouped according to average temperature, the Netherlands, 1979–1987. The size of the block is proportional to the square root of the corresponding number of days.

16.5°C temperature compares closely to a maximum temperature of 20.5°C). Average daily temperature was, therefore, measured by two complementary variables, "heat" (0 if the temperature is ≤16.5°C, temperature in degrees Celsius minus 16.5 if otherwise) and "cold" (0 if the temperature is ≥16.5°C, 16.5 minus temperature in degrees Celsius if otherwise).

Lagged effects of temperature were accounted for by including in the regression equation values of heat and cold for previous days. Ideally, the size of the effect would be assessed for each previous day individually; however, estimates of effects for separate days would be highly unstable because of the high correlation between temperature values for subsequent days. In order to reduce multicollinearity, temperature variables were constructed for groups of subsequent days (lag periods) by averaging the values for heat and cold over these periods.

The most effective way of grouping days into lag periods cannot be established on a priori grounds because of a lack of published material on this subject in the international literature. Therefore, a large number of combinations of lag periods were tried. All combinations were found to demonstrate a similar pattern of effects of temperature on mortality: large effects at less than 1 week, moderate effects at 1–4 weeks, and no significant effects after 1 month. The consistency of this observation suggests that it is not an artifact caused by multicollinearity. In this paper, results will be presented for a combination of lag periods which are able to represent the observed pattern economically: lag periods have an exponentially increasing size (1–2, 3–6, 7–14, and 15–30 days), while lag times of longer than 1 month are ignored.

A combination of lag periods reduces the multicollinearity problem but does not eliminate it entirely. Particularly high is the association between temperature values of the lag period 0 days (i.e., the same day) and the lag period 1–2 days ($r = 0.94$ for cold and 0.67 for heat). A number of ad hoc analyses were made which were specifically designed to avoid the potential effects of the high collinearity. For example, the relation between mortality and temperature on the same day was examined while restricting the

analysis to days with specific average temperatures (e.g., 10°C) only during the previous 2 days. The results of these analyses were found to be similar to those that will be presented below.

Regression coefficients β for individual lag periods were transformed by using the formula $100 * (\exp\beta - 1)$, to the percent change in mortality associated with a 1°C increase in the average value of cold or heat within the respective lag period ("percent effect"). Summing the transformed regression coefficients over individual lag periods yields an estimate of the percent change in mortality associated with a 1°C increase in the average value of cold or heat during the last month ("aggregate effect").

Control for influenza incidence, air pollution, and season was made by including these variables into the regression model. For influenza and air pollution, the same lag periods as for temperature were assumed so that control could be established for various time sequences (e.g., the effect of temperature on influenza incidence is rapid and the effect of influenza incidence on mortality is slow, or vice versa). The variable season was assumed to be constant between years and was measured by a 12-level nominal variable denoting the calendar months. Similar results were obtained with alternative ways of controlling for season: fitting a third order sinusoid curve to within-year mortality fluctuations or repeating the analysis for all January months only, all February months only, etc.

Effect modification by wind speed and relative humidity was examined by means of interaction (product) terms between cold and heat on the one hand and wind speed and relative humidity on the other. Since the number of interaction terms (four combinations of variables × five periods) is so large that it would not permit a meaningful presentation of each interaction term, results will be presented of an analysis which combined days 0–6 into one period and ignored the longer lag periods.

Regression models were fitted with the GLIM computer package (33). The significance of a regression coefficient was determined on the basis of the change in scaled deviance when the respective term was added to the regression model which included all the other terms.

RESULTS

A large portion of the daily mortality variation is associated with variation in temperature. When the variables cold and heat for five lag periods each are included in the regression model containing only the variables on age structure and long-term mortality trend, the scaled deviance is decreased sizably, from 9,759 to 4,969 (with 3,275 degrees of freedom remaining).

Distinction of lag periods

We will now examine whether most of the effects of cold and hot weather, respectively, occur within 1 week. In table 2, the first row under each Cold and Heat presents the results of the regression analysis without control for potential confounders.

In most lag periods, cold is positively related to the actual mortality level. The percent effect of 0.30 for the lag period 1–2 days means that a 1°C increase in the average value of cold over the 2 previous days (i.e., a 1°C decrease in average temperature below 16.5°C) is associated with a 0.30 percent mortality increase. The average temperature over the lag periods 3–6 and 7–14 days, respectively, has a larger effect. The effect for the lag period 15–30 days is relatively small. Thus, although a large part of the effect of cold on mortality occurs within 1 week, some of the effect occurs later.

A surprising finding concerns the lag period 0: The negative regression coefficient suggests that, with the temperatures in the preceding days being constant, cold is associated with lower mortality on the same day. The aggregate effect over all of the lag periods is 1.17 (table 2). This means that the actual mortality level is 1.17 percent higher for each degree Celsius increase in the average value of cold over the preceding month.

Hot weather has a large immediate effect on mortality on the same day and the 2

TABLE 2. Association between temperature and mortality, controlling for a number of variables, the Netherlands, 1979–1987

Temperature and control variable	% effect, by lag period†					Aggregate effect‡
	0	1–2	3–6	7–14	15–30	
Cold						
None	−0.25***	0.30***	0.44***	0.50***	0.18***	1.17
Influenza incidence	−0.27***	0.29***	0.42***	0.40***	−0.07	0.77
Sulphur dioxide density	−0.25***	0.29***	0.50***	0.58***	0.22***	1.34
Season	−0.26***	0.30***	0.43***	0.49***	0.22***	1.18
All 3 variables together	−0.27***	0.26***	0.45***	0.41***	0.06	0.91
Heat						
None	1.74***	1.24***	−0.22	−0.53***	−0.81***	1.42
Influenza incidence	1.77***	1.26***	−0.23	−0.56***	−1.14***	1.10
Sulphur dioxide density	1.77***	1.24***	−0.12	−0.45**	−0.69***	1.75
Season	1.72***	1.24***	−0.20	−0.47***	−0.43*	1.86
All 3 variables together	1.76***	1.23***	−0.14	−0.49***	−0.51*	1.85

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.
† Percent mortality increase per change in degrees Celsius. (The effect of 1 degree of change during 1 individual day within a lag period is about equal to the effect for the entire period divided by the number of days.) Estimated from regression analysis of mortality on the average values of "cold," "heat," and the respective control variables in the five lag periods. Regression coefficients are transformed according to the formula in Materials and Methods.
‡ The sum of the percent effects associated with the five lag periods.

subsequent days, as shown by the positive association for the lag periods 0 and 1–2 days. Longer lag periods show a compensatory effect: Heat 7–30 days before is inversely, instead of positively, related to the actual mortality level. The negative effects after longer lag periods (0.22 + 0.53 + 0.81 = 1.56) compensate for about half of the positive effect within 3 days (1.74 + 1.24 = 2.98).

Control for influenza incidence, air pollution level, and season

The relation between mortality and cold is altered very little by control for sulphur dioxide density and season (table 2). Control for influenza incidence diminishes the aggregate effect by 34 percent (1 − 0.77/1.17) by explaining a small part of the effect of cold 7–14 days previously, and the entire effect of cold more than 14 days previously.

The relation between heat and mortality is about one third explained by increased incidence of influenza-like conditions, which mainly alters the effect of heat which occurred 2 or more weeks previously. Control for season does not diminish the aggregate effect; instead, this factor seems to have obscured some of the heat-related mortality. An explanation for this is that the occurrence of hot weather is strongly associated with the summer season, when, in turn, mortality levels are generally low (34). Control for sulphur dioxide density does not diminish the aggregate effect. The same result was found in an analysis which used data on components of photochemical smog such as ozone and nitrogen dioxide (results not shown here).

Effect modification by wind speed and humidity

We will now examine whether the effect of cold is greater when cold temperature is accompanied by strong winds and whether the effect of heat is greater when heat is accompanied by weak winds and high relative humidity. Figure 2 shows that the effect of cold is indeed enhanced by strong winds, but is also enhanced by high relative humidity. The interaction of cold with both factors is significant ($p < 0.001$).

Heat has a statistically significant interaction ($p < 0.001$) with relative humidity but not with wind speed. Figure 2 shows that the effects of heat are diminished, as opposed to increased, by humid weather. The hypothesized pattern of stronger effects of heat during humid weather was not observed by graphic inspection, cause-specific analysis, or examination of absolute rather than relative humidity.

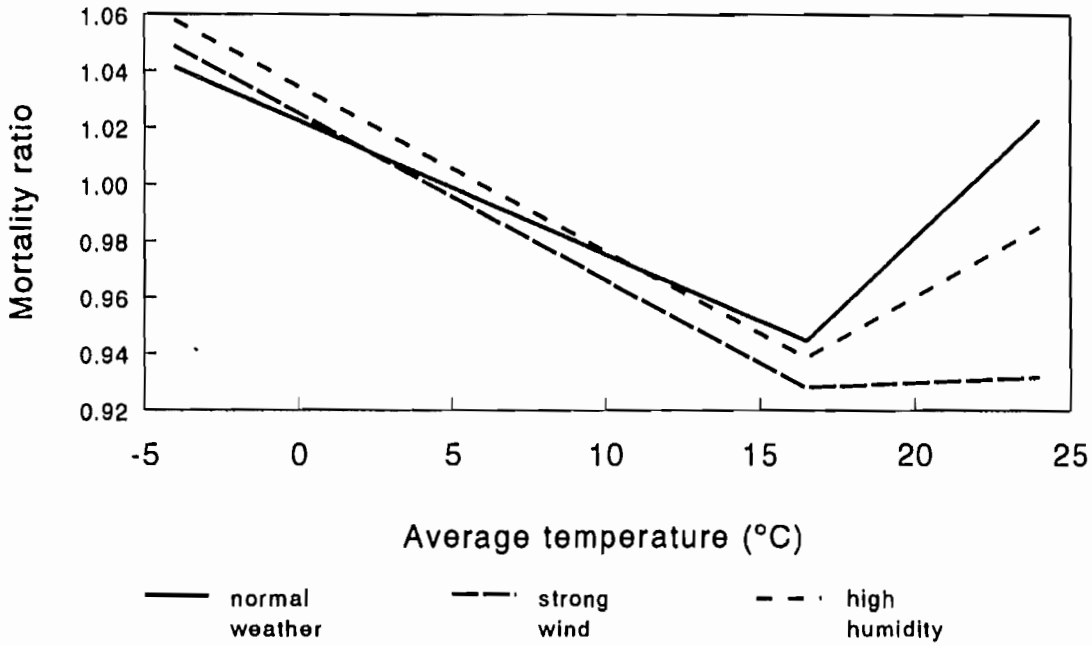

FIGURE 2. The association between mortality and temperature in the lag period 0–6 days, with high relative humidity, strong winds, and normal weather conditions, respectively, the Netherlands, 1979–1987. Values are derived from the regression analysis described in the Materials and Methods section. The mortality ratios by temperature, wind speed, and relative humidity (with the overall average being 1.00) were estimated from the regression equation by filling in the estimated regression coefficients and values for temperature, wind speed, and relative humidity. Normal wind = 2.85 m/second, strong wind = 4.10 m/second (one standard deviation higher); normal relative humidity = 79.2%, high relative humidity = 88.3% (one standard deviation higher). ——, normal weather; — —, strong winds;– – –, high relative humidity.

Distinction of causes of death

Daily variation in cause-specific mortality is significantly associated with variation in temperature levels. For cardiovascular and respiratory disease mortality, as for all-cause mortality, inclusion of the variables cold and heat for five lag periods in the regression model causes about a 50 percent decrease in scaled deviance. The reduction in scaled deviance for other groups of causes of death is smaller (between 10 and 20 percent) but is also statistically significant.

Regression estimates per lag period are presented in table 3 for main groups of causes of death. These estimates are based on the model which includes all three control variables. Cold in preceding days is positively related to all causes except for external causes. The effect of cold on cardiovascular diseases is slightly greater than for total mortality, but much weaker than for respiratory disease mortality. In the last column of table 3, the absolute number of cold-related deaths is estimated at 2.95 deaths per day for every degree Celsius increase in the average value of cold over the last month. Of these deaths, 57 percent (1.68/2.95) is caused by cardiovascular diseases.

Heat has a rapid positive effect on all causes of death, and particularly on respiratory diseases and external causes of death. The effect on cardiovascular disease mortality is not larger than the effect on total mortality. After longer lag periods, strong compensatory effects occur for neoplasms and cardiovascular diseases, but not for other diseases. Cardiovascular diseases are responsible for only 26 percent (1.54/6.01) of all heat-related deaths.

Analysis of residuals

Variation in residuals is larger than could be expected from Poisson variation alone. An example is the regression of total mor-

TABLE 3. Relation between temperature and mortality by cause of death, the Netherlands, 1979–1987

Temperature and cause of death	% effect, per lag period†					Aggregate effect‡	
	0	1–2	3–6	7–15	15–30	Relative	Absolute
Cold							
All causes	−0.27***	0.26***	0.45***	0.41***	0.06	0.91	2.95
Neoplasms	−0.56***	0.11	0.51***	0.11	−0.31	−0.14	−0.12
Cardiovascular diseases	−0.02	0.33***	0.37***	0.46***	0.03	1.17	1.68
Respiratory diseases	−0.55**	0.31	0.54*	1.43***	0.92**	2.65	0.43
All other diseases	−0.31**	0.34*	0.65***	0.43***	0.39*	1.50	0.90
External causes	−0.60***	0.06	−0.13	0.35	0.21	−0.11	−0.02
Heat							
All causes	1.76***	1.23***	−0.14	−0.49***	−0.51*	1.85	6.01
Neoplasms	1.33***	0.80**	−1.08***	−0.91**	0.03	0.17	0.15
Cardiovascular diseases	1.75***	1.13***	−0.17	−0.75**	−0.89**	1.07	1.54
Respiratory diseases	3.31***	3.11***	3.08***	1.73*	−0.79	10.44	1.70
All other diseases	1.79***	1.56***	0.30	0.09	−0.33	3.41	2.05
External causes	2.46***	1.28*	0.62	−0.19	−0.75	3.42	0.55

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

† Percent mortality increase per change in degrees Celsius. Estimated from regression analysis of mortality on the average values of both "cold" and "heat" in the five lag periods, controlling for influenza incidence, sulphur dioxide density, and season. Regression coefficients are transformed according to the formula in Materials and Methods.

‡ The relative aggregate effect is calculated as the sum of the percent effects associated with the five lag periods. The absolute aggregate effect is calculated as the relative aggregate effect times the average daily number of deaths (as given in table 1). The estimate for total mortality is not equal to the sum for separate causes of death (2.87 for cold and 5.99 for warm) because of random error in regression estimates.

tality on temperature and the three control variables (table 3). The residual scaled deviance was 4,041 as opposed to 3,254 degrees of freedom (which is equal to the expected scaled deviance under the Poisson distribution). Inspection of residuals did not show extreme outliers. Nonrandom residual variation suggests that not all potential confounders have been included in the regression model. The implications of this will be discussed below.

Residuals were found to have a weak autocorrelation. In the example above, autocorrelation for the lag period 1 day was 0.12. Although autocorrelation implies that confidence intervals are in fact wider than is suggested by the outcomes of the regression analyses (35), it is so weak that a correction for it would not sizably enlarge the small p values found in this study.

DISCUSSION

Man is a tropical animal (8, 36). At moderate heat he feels most comfortable (37). Exposure to higher or lower ambient temperatures causes stress on the circulatory and other body systems. This paper deals with the question of whether this stress mechanism is directly responsible for the well-known fact that death rates become progressively higher when outdoor air temperatures get hotter or colder than 20–25°C. The findings for cold- and heat-related mortality will be discussed separately.

Cold-related mortality

Our findings support the idea that the relation between cold weather and mortality is largely attributable to the direct effects of exposure to cold temperatures. Approximately one third of cold-related mortality can be explained by the indirect effects of influenza, air pollution, and season. However, it cannot be concluded that the unexplained two thirds of cold-related mortality can be attributed to direct effects because not all potential confounders could be included in this analysis. The presence of unidentified confounders is suggested by the nonrandom variation in residuals. However, further support for the hypothesis that the cold temperature-mortality relation is due to direct effects is that the unexplained cold-related mortality occurs rapidly (within 1 week) and is greater if cold temperatures are accompanied by strong winds.

Our results show that a portion of cold-related mortality results from the indirect effect of an increased incidence of influenza or influenza-like conditions. The contribution of influenza to cold-related mortality has been discussed frequently (4, 5, 14–17), but has not been assessed empirically. Our analysis shows that influenza or influenza-like conditions explain about one third of the excess mortality after periods of cold weather. The finding that long-term (more than 1 week), but not the short-term, effects of cold weather on mortality are thus explained is suggestive of a gradual causal-time sequence in which periods of cold weather give rise to an increased incidence of influenza or influenza-like conditions which, in turn, result in higher death rates. Impaired immunologic defense might play a role, although evidence concerning this mechanism is weak (38).

It has been argued that respiratory infections are also responsible for the cold-related deaths for which the underlying cause has been coded as cardiovascular disease (4, 14–17). However, this idea is inconsistent with the finding that most cold-related cardiovascular deaths occur within a few days of the cold period. Moreover, while respiratory infections are expected to affect mainly case fatality due to cardiovascular diseases, both case-fatality and incidence rates rise after cold weather (5, 14, 26, 39, 40).

As direct effects of cold on the human body seem to play an important role, a subsequent question is to what extent these effects can be attributed to increased cardiovascular stress. The fact that cardiovascular diseases are the underlying cause of about half of all cold-related deaths that are not explained by increased influenza incidence suggests that cardiovascular stress plays an important role. However, additional mechanisms are suggested by the fact that a large number of cold-related deaths are the result of other diseases and that the relative effect of cold weather is greater on respiratory diseases than on cardiovascular diseases. A possible mechanism is that breathing cold air leads to bronchoconstriction, which might increase susceptibility to, or worsen, pulmonary infections (41, 42).

In conclusion, it seems that a large portion of cold-related mortality is attributable to the direct effects of exposure to cold on the human body, but only partially by means of increased stress on the circulatory system.

Previous studies have suggested that part of the cold-mortality relation is merely an artifact due to confounding by season (14, 19–21). For example, Bowie and Prothero (20) noted that correction for seasonal mortality variation diminishes the correlation between temperature and mortality. However, our study showed that when season is treated in the same way that confounders are commonly treated (i.e., not by a priori adjustment, but by simultaneous inclusion into the regression equation), this factor has a negligible effect on the cold-mortality relation. As reported elsewhere (32), we found that the effect of season on mortality is modest when controlled for temperature and influenza incidence.

A new and quite surprising finding is that cold weather is associated with lower instead of higher mortality during the same 24-hour period. This result could not have been obtained in previous studies because the relation between cold weather and mortality during the same 24-hour period has never been analyzed while controlling for temperature on previous days. We feel that speculation about the possible causes will be meaningful only when this finding has been replicated in other studies.

Heat-related mortality

This study focused on the mortality effects of moderate heat; therefore, the results cannot be directly compared with the results of other studies which, without exception, were concerned with periods of excessive heat (43–46).

Our findings are in agreement with the idea that heat-related mortality is largely attributable to the direct effects of exposure of the human body to hot ambient air. The effect of hot weather on mortality can to only a modest extent be explained by influenza-like conditions, air pollution, and season. In addition, most of the unexplained effect of hot weather on mortality occurred rapidly.

An unexpected finding is that high humidity diminishes, instead of enhances, the effects of hot weather. Enhanced effects were expected because high humidity reduces evaporation of sweat and in this way impairs body cooling (8). This idea also underlies the construction of several indices of thermal comfort (25). Our results indicate that the role of sweating has probably been exaggerated. Thermophysiologic laws state that for naked persons at rest, sweating is needed when ambient air temperatures exceed approximately 30°C (47). In the general population, which is clothed and physically active, the threshold temperature may be lower. However, our findings suggest that in a temperate climate this threshold is surpassed by a significant extent on only a few days. Consistent with this explanation are the findings of an analysis in the United States that relative humidity modifies the effects of temperature on mortality only in the hottest regions (48).

Indications were found of an indirect mechanism involving an increased incidence of influenza-like conditions. As is true with cold-related mortality, influenza-like conditions seem to be responsible for some of the long-term (more than 1 week), but not for the short-term, effects of hot weather on mortality. The possibility that heat has a causal effect on the incidence of influenza or influenza-like conditions has to our knowledge received little attention in the literature.

Since a large portion of heat-related mortality seems to be attributable to the direct effects of exposure of the human body to air, a subsequent question is to what extent these effects are the result of heat-induced cardiovascular stress. The findings that only about a quarter of all heat-related deaths are the result of cardiovascular diseases and that the relative effect of heat on cardiovascular disease mortality is much smaller than on some other disease categories suggest that the role of heat-induced cardiovascular stress is very modest.

A more important factor is suggested by the especially large increase in respiratory disease mortality, even after control for influenza incidence. The fact that large effects occur within 1 week should be attributed to a rapidly evolving mechanism. The finding that humid weather is protective against the effects of heat suggests that the inhalation of dry and hot air is harmful to the respiratory organs, perhaps because of the presence of irritants.

Although hot weather has a large immediate effect on cardiovascular mortality, this effect is largely compensated for by lower mortality in subsequent weeks. This finding strongly suggests a "harvesting effect": Heat takes its toll principally among terminal patients whose death would occur anyhow within a few days or weeks. The reason terminal patients succumb during hot weather is unknown; it might be related to cardiovascular stress because of exposure of the human body to heat. However the mechanism operates, the small amount of living time lost per death makes this factor a public health problem of limited importance.

In conclusion, there are strong indications that a large portion of heat-related mortality is attributable to the direct effects of exposure to heat on the human body, but that increased cardiovascular stress is not an important contribution.

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Anonymous
MMWR. Morbidity and Mortality Weekly Report; Jul 4, 2003; 52, 26; ProQuest
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The findings in this report are subject to at least five limitations. First, only nonfatal injuries treated in hospital EDs were included, and injuries treated in health-care facilities outside of an ED (e.g., a physician's office or an urgent care center) or those for which no care was received were not included. Previous estimates indicate that 17% of dog bite–related injuries are treated in medical facilities, of which 38% are seen in hospital EDs (*1*). Second, injury diagnoses were not specified for 26% of cases. Third, limited data are available on the circumstances of the event or the dog involved. Fourth, NEISS-AIP is designed to provide national estimates and does not provide state or local estimates. Finally, although the extent of human exposure to dogs might vary by age, sex, season, or other factors, these data are not available; as a result, the analysis did not account for exposure.

Prevention programs should educate both children and adults about bite prevention and responsible pet ownership. Additional information about preventing dog bites is available at http://www.cdc.gov/ncipc/duip/dogbites.htm.

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Heat-Related Deaths — Chicago, Illinois, 1996–2001, and United States, 1979–1999

Heat waves (i.e., ≥3 consecutive days of air temperatures ≥90° F [≥32.2° C]) are meteorologic events that contribute significantly to heat-related deaths. Exposure to excessive heat can cause illness, injury, and death. This report describes four cases of heat-related deaths*, as reported by the Office of the Medical Examiner, Cook County, Chicago, that occurred during 1996–2001; summarizes total heat-related deaths in Chicago during 1996–2001; and compares the number of heat-related deaths during the 1995 and 1999 Chicago heat waves. This report also summarizes trends in the United States during 1979–1999, describes risk factors associated with heat-related deaths and symptoms, and outlines preventive measures for heat-related illness, injury, and death. Persons at risk for heat-related death should reduce strenuous outdoor activities, drink water or nonalcoholic beverages frequently, and seek air conditioning.

Case Reports

Case 1. In June 1997, a woman aged 86 years with no known medical history was found unresponsive in her bedroom. Her grandson reported that the woman had kept the bedroom windows closed for a week and that the room was very hot. The room had no fan. Paramedics transported the woman to the hospital, where a rectal temperature of 108° F (42.2° C) was recorded. She was pronounced dead in the emergency department. An autopsy revealed moderate coronary atherosclerosis. Heat stroke was listed as the cause of death, with arteriosclerotic cardiovascular disease as a significant contributing condition.

Case 2. In July 1999, a woman aged 73 years whose medical history was unknown was found unresponsive behind a building. She had been seen earlier in the day drinking alcohol. Paramedics transported her to the hospital, where she was pronounced dead on arrival.

Her rectal temperature was registered as 108° F (42.2° C). An autopsy revealed a blood alcohol level of 117 mg/dL (legal blood alcohol limit in Illinois is 80 mg/dL) and a vitreous alcohol level of 157 mg/dL. The cause of death was listed as heat stroke.

* Defined as one in which exposure to high ambient temperatures either caused the death or contributed to it substantially, body temperature at the time of collapse was ≥105° F (≥40.6° C), the decedent had a history of exposure to high ambient temperature, and other causes of hyperthermia could reasonably be excluded (*1*). Because rates of death from other causes (e.g., cardiovascular and respiratory disease) increase during heat waves (*2,3*), deaths classified as caused by hyperthermia represent only a portion of heat-related mortality.

Case 3. In March 2000, a man aged 35 years was found unresponsive in a steam room at a health club. Before entering the steam room, the man reportedly had slurred speech, and his hands were shaking. He was transported to the hospital, where he was pronounced dead on arrival. An autopsy documented previous hypertensive cerebral damage and a recent right basal ganglia infarct. The cause of death was listed as hyperthermia caused by hypertensive cardiovascular disease.

Case 4. In September 2000, a girl aged 5 months was found unresponsive in the back seat of her parents' car by her mother. The child had been left mistakenly in the car for 9 hours while her parents were at work. Paramedics transported the child to the hospital, where she was pronounced dead on arrival. The medical examiner listed the cause of death as heat stroke.

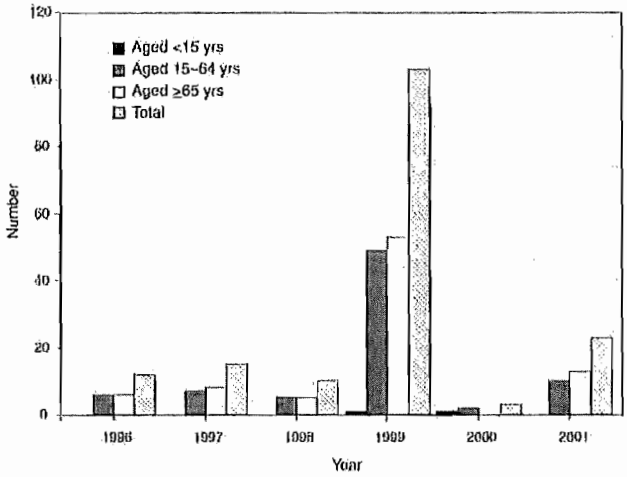
Summary of Heat-Related Deaths in Chicago and U.S. Trends

During the 1990s, Chicago experienced two heat waves. In July 1995, a heat wave resulted in 485 heat-related deaths and 739 excess deaths (*4*). An epidemiologic investigation of the heat wave identified advanced age and an inability to care for oneself as major risk factors for heat-related death (*5*). During 1999, a heat wave resulted in 103 heat-related deaths; 80 were attributed to extreme heat. Implementation of Chicago's Extreme Weather Operations Plan reduced the death toll by increasing the number of daily contacts for the elderly during the 1999 heat wave (*6*). During 1996–2001, annual totals of heat-related deaths across all age groups was highest in 1999 (Figure 1).

During 1979–1999, the most recent years for which national data are available, 8,015 deaths in the United States were heat-related†. A total of 3,829 (48%) were "due to weather conditions," 377 (5%) were "of man-made origins" (e.g., heat generated in vehicles, kitchens, boiler rooms, furnace rooms, and factories), and 3,809 (48%) were "of unspecified origin" (*7*). An average of 182 deaths per year (range: 54–651) were associated with excessive heat resulting from weather conditions. Of the 3,764 (98%) weather-related deaths for which age of decedent was reported, 1,891 (49%) occurred among persons aged 15–64 years, 1,709 (45%) occurred among persons aged ≥65 years, and 164 (4%) occurred among children aged <15 years (*7*). During 1979–1999, rates for heat-related deaths increased with age (Figure 2).

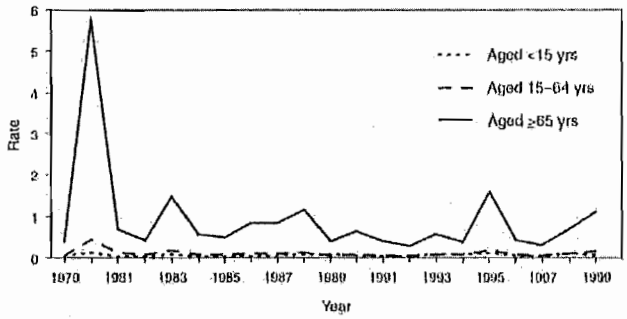
† During 1979–1998, the underlying cause of death attributed to excessive heat exposure was classified according to the *International Classification of Diseases, Ninth Revision* (ICD-9), code E900.0, "due to weather conditions"; code E900.1, "of man-made origins"; and code E900.9, "of unspecified origin." Data for 1999 were obtained from ICD-10; code X30, "exposure to excessive natural heat (deaths)," was added to the 1979–1998 ICD-9 code E900.0, "excessive heat due to weather conditions (deaths)." Data were obtained from the Compressed Mortality File of CDC's National Center for Health Statistics, which contains information from death certificates filed in the 50 states and the District of Columbia.

FIGURE 1. Annual totals of heat-related deaths attributed to weather conditions* and exposure to excessive natural heat†, by age group — Chicago, 1996–2001



* *International Classification of Diseases, Ninth Revision* (ICD-9), code E900.0.
† ICD-10, code X30.

FIGURE 2. Average annual rate* of heat-related deaths attributed to weather conditions† and exposure to excessive natural heat§, by age group and year — United States, 1979–1999



* Per 100,000 population.
† *International Classification of Diseases, Ninth Revision* (ICD-9), code E900.0.
§ ICD-10, code X30.

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Editorial Note: Exposure to high temperature for a sustained period can cause heat-related illness (hyperthermia) or death. The two most serious types of heat-related illness are heat

exhaustion and heat stroke. Heat exhaustion is characterized by paleness, fatigue, muscle cramps, dizziness, headache, nausea or vomiting, and fainting. The skin is typically cool, and moistness and sweating might occur. The pulse rate is fast and weak, and breathing is fast and shallow. If untreated, heat exhaustion can progress to heat stroke (*8*). Heat stroke is a serious, often fatal condition characterized by a body temperature of >103° F (>39.4° C); red, hot, and dry skin (no sweating); rapid, strong pulse; throbbing headache; dizziness; nausea; confusion; and unconsciousness.

Elderly persons, children, and persons with certain medical conditions (e.g., heart disease) are at greatest risk for heat-related illness and death. Drinking alcohol, participating in strenuous outdoor physical activities in hot weather, and taking medications that reduce the body's ability to regulate its temperature or that inhibit perspiration also increase risk. Air conditioning is the most important protective factor against heat-related illness and death. For the 1995 and 1999 Chicago heat waves, the risk for heat-related death increased for persons with cardiac disease or psychiatric illness and for persons who lived alone. Having a working air conditioner and participating in group activities in which heat-related illness might be identified were the most important protective factors (*5,6*) (Box).

Heat-related morbidity and mortality could increase with periods of extreme heat (*9*). Many cities have developed emergency-response plans for heat waves. These response plans use information about risk factors and meteorologic conditions to implement prevention strategies that reduce morbidity and mortality from excessive heat (*10*). A heat-response plan also should recommend rolling energy blackouts in areas that use air conditioning to mitigate factors that increase the risk for heat-related morbidity and mortality. To defray energy costs, support of low-income populations might be necessary to allow the use of air-conditioning during summer months.

BOX. Measures for preventing heat-related deaths

During heat waves
- Check on elderly, disabled, or homebound persons frequently.
- Never leave children alone in cars and ensure that they cannot lock themselves in an enclosed space (e.g., a car trunk).
- Evaluate persons at risk for heat-related death frequently for heat-related hazards and illnesses, and take appropriate preventive action.
- Seek air-conditioned environments.

If exposure to heat cannot be avoided
- Reduce, eliminate, or reschedule strenuous activities.
- Drink water or nonalcoholic fluids frequently.
- Take showers regularly.
- Wear light-weight and light-colored clothing.
- Avoid direct sunlight.

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Multistate Outbreak of *Salmonella* Serotype Typhimurium Infections Associated with Drinking Unpasteurized Milk — Illinois, Indiana, Ohio, and Tennessee, 2002–2003

On December 10, 2002, the Clark County Combined Health District and the Ohio Department of Health (ODH) were notified of two hospitalized children infected with *Salmonella Enterica* serotype Typhimurium. Initial investigation implicated consumption of raw, unpasteurized milk purchased at a local combination dairy-restaurant (dairy) during November 27–December 13, 2002, as the cause. This report summarizes the subsequent investigation. Because 27 states still allow the sale of raw milk, and organizations continue their efforts to allow marketing and sale of raw milk to the public directly from the farm (*1,2*), consumer education about the hazards of raw milk and a careful review of existing policies are needed.

The dairy comprised a working dairy farm, restaurant, snack bar, and petting zoo with goats, cows, calves, lambs, and pigs. At the time of the epidemiologic investigation in December 2002, the workforce comprised 211 workers, including 16 members of the owner family. In 2002, the dairy was the only place in Ohio that sold raw milk in jugs and served raw milk and milk shakes made with raw milk legally to customers. In 2001, approximately 1,350,000 customers visited the dairy.

During November 30, 2002–February 18, 2003, ODH laboratory received 94 *S.* Typhimurium clinical isolates for pulsed-field gel electrophoresis (PFGE) testing. Of these, 60 had an indistinguishable pattern. In addition, patterns from Illinois, Indiana, and Tennessee matched the Ohio pattern.

A case of *S.* Typhimurium was defined as PFGE–matched *S.* Typhimurium isolated during November 30, 2002–February 18, 2003, from clinical samples from a person with an epidemiologic link to the dairy. Case finding was conducted by reviewing laboratory culture results from hospital, private, and ODH laboratories, comparing PFGE patterns of *S.* Typhimurium isolates with background isolates statewide and nationwide, screening dairy workers, interviewing meal companions, and alerting public health officials of the outbreak nationwide by using CDC's *Epidemic Information Exchange* (*Epi-X*).

A total of 62 persons had illness consistent with the case definition, including 40 customers, six household contacts, and 16 (7.6%) of 211 dairy workers; patients were from four states (Illinois, Indiana, Ohio, and Tennessee); the median age was 18 years (range: 1–70 years), and 34 (54.8%) were females. Of the 62 patients, 54 (87.1%) reported signs and symptoms of illness, including diarrhea (52 [96.3%]), cramps (41 [75.9%]), fever (37 [68.5%]), chills (29 [53.7%]), body aches (29 [53.7%]), bloody diarrhea (27 [50.0%]), nausea (25 [46.3%]), vomiting (24 [44.4%]), and headache (21 [38.9%]). A total of 50 (80.6%) exhibited more than one symptom. Disease onset occurred during November 30, 2002–January 14, 2003 (Figure).

A case-control study was conducted to verify the initial findings implicating raw milk and to identify other potential sources of infection. The 40 case-patients who were dairy customers were included in the study. Controls were a convenience sample of well meal companions of case-patients. Because of numerous potential exposures to *S.* Typhimurium, dairy workers were excluded from the study; secondary infections among friends or households contacts of case-patients also were excluded. Food histories were obtained through telephone interviews by using a standard questionnaire. State and local investigators reviewed milking, bottling, and capping procedures and collected and tested samples from the food, stools of dairy cows, and the environment.

A total of 40 case-patients and 56 controls were eligible for the case-control study. The median age of case-patients was 8 years (range: 1–69 years); 24 (60.0%) were females. The

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ENVIRONMENT, TEMPERATURE AND DEATH RATES

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Summary

Analysis of recorded monthly deaths in England and Wales shows a close association of death rates with external temperature in most diseases other than the cancers.

Analysis of daily deaths in England and Wales and in New York shows the following relationships between temperature and deaths from myocardial infarction, strokes and pneumonia. Between −10° and +20°C minimum temperature there is a nearly linear fall in deaths as the temperature rises. Above 20°C deaths rise steeply as the temperature rises and below −10°C rise steeply as temperature falls. These associations of deaths with temperature are much stronger in the elderly than in younger subjects.

Detailed analysis of the daily deaths in England and Wales from myocardial infarction, strokes and pneumonia show that short-term (1–2 days) temperature changes have little effect on death rates but medium-term (7–10 days) and longer-term (three or more weeks) changes are associated with very significant changes in death rates.

The three diseases vary in the time relations between temperature change and change in death rates. In all three there is an interval between the change in temperature and death and this is shortest in the case of myocardial infarction (1–2 days before death), longest in the case of pneumonia (about a week before death) and intermediate in the case of strokes (about 3–4 days before death).

At low temperatures death rates increase as the duration of temperature change increases, while at high temperatures (but below +20°C) death rates decrease as the period of temperature change is longer.

The implications of these findings are discussed and it is postulated that there is probably a causal relationship between temperature change and deaths from a wide variety of diseases. A proximal link in the chain is probably a failure of autonomic control of body temperature in the elderly leading to a change in body temperature and some humoral change which in turn leads to death. It is not appropriate to concentrate on hypothermia as the relationship between temperature and death is seen at all temperatures.

Introduction

In previous publications we have reported on the relationships between atmospheric temperature and death rates from all causes combined and from certain respiratory and arteriosclerotic disease. We have shown that in England and Wales, especially in older subjects, changes in temperature are associated with inverse changes in death rates from these conditions (Bull 1973, Bull & Morton 1975*a* and *b*).

It is tempting to speculate that the changes in death rates are caused by changes in temperature and we have explored this possibility in a number of ways. One was to seek for associations between ambient temperature and the levels of a wide variety of blood constituents in the hope that this might reveal possible ways in which temperature might affect death rates. No significant relationships emerged with commonly estimated chemical or haematological variables but the study revealed a weak but statistically

significant relationship of temperature with factor VII, antithrombin III and fibrinolytic activity levels (Bull et al. 1978 unpublished).

Another approach was to explore the relationship of temperature with death rates from a larger variety of diseases. A third was to analyse data from New York where the range of ambient temperature is much wider than in Britain, and a fourth was to define more exactly the interaction between age and duration of temperature change with death rates from myocardial infarction, cerebral vascular accidents and pneumonia. These last three approaches form the burden of this paper.

Materials and Methods

The data on which the investigation of the relationship between temperature and death rates from a variety of diseases is based are from the Registrar General's Statistical Review of England and Wales for the years 1963 to 1966 (tables 22 and 34), supplemented by temperature and population data from 1962 to 1967. The figures for deaths by month were corrected to a standard 30-day month and to a population based on the mid-1963 figure. They were then averaged and each month's figure was calculated as a percentage of that average. Standard statistical techniques were used to calculate the coefficient of variation of deaths (i.e. standard deviation of deaths per month expressed as a percentage of the mean monthly death rate). From analyses of variance the percentages of total variance associated with mean monthly temperature and the slopes of regressions of death rates on temperature were also calculated.

The data for the rest of the studies were from two sources. The New York data were kindly supplied by the Bureau of Health Statistics and Analysis, City of New York Department of Health, and we are indebted to Mrs Frieda Nelson of that Department for arranging this. They consisted of individual records of all deaths from myocardial infarction (ICD 420.1) and strokes (ICD 330–334) in New York during the period 1 January 1965 to 31 December 1968. Each record gave the patient's sex, age and date of death. Temperature data for New York were kindly provided by the Environmental Data Service, National Climatic Center, Asheville, North Carolina.

The data for England and Wales were in the same form and provided by the Registrar General for England and Wales for the years 1970 and 1971. The temperature data relate to London Airport.

Figs. 1–5 and associated Table II were calculated as follows. Although temperatures were recorded to 0.1 °C, each day's temperature was transformed to the nearest integer. In this way the total temperature range was reduced to equal divisions of width 1 °C. The average number of deaths per day the temperature was within each division of width 1 °C was then calculated and plotted against temperature. Standard statistical techniques were used to calculate the data shown in Table I and the regression lines in the figures. In the case of the New York data the calculations were made in three ranges of temperatures: less than −10 °C, −10 to +20 °C and above 20 °C.

The techniques used in the next part of the study have been reported in detail elsewhere (Bull & Morton 1975*a* and *b*) but are in summary as follows.

Having shown that there was no significant difference between males and females in respect of the relationships between temperature and deaths, the analyses presented relate to both sexes combined. Separate tables were prepared for the age groups <60 and 60+ giving the daily deaths and temperatures arranged as below. (In the case of pneumonia all days on which the temperature was below −4 °C were excluded because of the absence of linearity of the deaths on temperature regression in the few days of very low temperature.)

Days	−2	−1	1	2	3	4	5	6	730
No. deaths			n_1	n_2	n_3	n_4	n_5	n_6	n
Temperatures	t_{-2}	t_{-1}	t_1	t_2	t_3	t_4	t_5	t_6	t

Then, in order to test the duration of temperature change necessary to produce or be associated with a change in death rates, the following procedure was adopted.

For 'Groups of days 1', $\sqrt{(n_2-n_1)}$, $\sqrt{(n_2-n_2)}$, etc. were correlated with (t_2-t_1), (t_3-t_2), etc.

For 'Groups of days 2', $\sqrt{[(n_4+n_3)-(n_2-n_1)]}$, etc. were correlated with $(t_4+t_3)-(t_2+t_1)$, etc.

For 'Groups of days 3', $\sqrt{[(n_6+n_5+n_4)-(n_3+n_2+n_1)]}$ were correlated with $[(t_6+t_5+t_4)-(t_3+t_2+t_1)]$, etc., similarly for higher groups of days up to 20.

In order to test whether the temperature on the days of death or on some preceding day or days was more relevant, the temperature figures one or more days to the left were used. Thus to determine the relevance of the temperature one day before death t_{-1} corresponded to n_1, t_1 to n_2, etc. Further shifts tested days -2, -3, etc. This method can also be seen as a method of testing for a phase shift between the curve of deaths and that of temperatures. It proved remarkably sensitive.

From the values for the correlation coefficients, calculations were made of the percentages of the variances associated with temperature from the formula $100r^2$ = percentage variance associated with temperature. [The residual variances are $100(1-r^2)$.]

From the tables produced in this way were plotted two-dimensionally what might be termed 'isovariances' which are equivalent to contour maps (the left-hand of the figure). On the right of each figure the probability values are plotted in the same way as what might be termed 'isoprobs'. In the middle of the figure is a scale of groups of days which applies to both sides of the figure. On the extreme left of each figure is a column of figures tabulating the coefficient of variation of deaths (S.D.%). Beside the figures is a key to the shading. On the left-hand side is a column of figures showing the percentage variance accounted for by temperature and on the right the probability.

The last part of this analysis concerns the effect of duration of temperature change on death rates. In the previous part of this analysis groups of days were formed by simple summation of deaths and temperatures as above. The sums therefore include periods where the temperatures might vary fairly widely and the analysis does not reveal very clearly the effect of runs of days when the temperature remained much the same. To show the effect of duration of 'runs of days', the same data were retabulated as follows. First the daily temperatures were classified into five classes—<0.1, 0.1–4.0, 4.1–8.0, 8.1–12.0, >12.0. Then the 730 days of data were classified into 'runs of days' in which the temperature remained within any of the five classes for 6 or more days, 5, 4, 3, 2 days or 1 day. Average numbers of deaths for each subclass were then calculated, excluding from each lower class of 'runs of days' data from any higher class. Finally regressions were calculated of the deaths per day on 'runs of days' classes for each temperature class. The results are shown in Table III.

Results

Temperature relationships of death rates in a variety of diseases

Table I shows the degree of association between mean monthly temperature and death rates from a variety of causes.

Comparisons between England and Wales and New York

The results are shown in Figs 1–5 and Table II. Note that in the middle range of temperatures in New York (-10 to $+20$°C) and in the total range of England and Wales, the regression of deaths on temperature is linear or nearly so.

Age, 'groups of days' and temperature phase shift effects

These are shown in Figs 6–11: their construction is explained under *Methods*. Note the figures in the left-hand columns (S.D.%) which are the values of the coefficient of variation for each 'group of days' shown in the middle column. The coefficients of variation are highest in the case of pneumonia (shown here simply as an example of a respiratory group of diseases) but are also substantial in the case of myocardial infarc-

Table I. Relationships between death rates and mean temperatures (1963–6) in England and Wales

ICD	*Diagnosis*	*Male*			*Female*		
		S.D.D.%	*B*	*VAT* %	S.D.D.%	*B*	*VAT* %
0	All causes	18.7	−3.4	82	20.0	−3.6	79
490	Lobar pneumonia	48.7	−8.3	72	53.0	−9.1	72
500	Acute bronchitis	77.5	−13.0	69	87.2	−14.7	70
502	Chronic bronchitis	49.8	−8.9	79	59.0	−10.3	75
241	Asthma	20.1	0.8	4	14.0	0.3	1
540	Gastric ulcer	44.1	−4.4	25	34.2	−4.4	41
541	Duodenal ulcer	31.0	−5.3	72	33.1	−5.6	71
550–553	Appendicitis	23.4	−2.3	24	22.3	−1.0	4
330–334	Strokes	19.7	−3.6	83	19.3	−3.5	80
420	Ischaemic heart disease	15.0	−2.8	83	17.8	−3.2	82
440–447	Hypertension	27.0	−4.7	73	32.2	−3.5	29
460–466	Diseases of veins	16.9	−2.4	52	16.6	−2.6	60
140–239	Neoplasms	5.2	−0.6	37	4.6	−0.6	37
151	Carcinoma—stomach	3.9	−0.4	30	5.8	−0.6	24
152–154	intestine and rectum	5.7	−0.5	17	5.1	−0.4	16
162–165	lung, bronchus	6.8	−0.8	31	9.9	−0.3	2
170	breast				6.9	−1.0	49
204	Leukaemia	28.3	−0.7	1	27.6	−0.5	0
590–637	Genito-urinary diseases	13.8	−2.2	65	11.8	−1.9	64
610	Hyperplasia of prostate	18.2	−2.8	7			
750–759	Congenital malformation	9.2	−1.2	41	10.3	−1.4	45
751	Spina bifida and meningitis	26.2	−2.5	22	20.8	−0.8	4
800–999	Accidents and violence	5.2	−0.6	38	14.3	−2.7	89
810–825	Accidents—motor vehicle	14.2	0.3	1	18.7	−0.1	0
970–973	Suicide by poisoning	12.5	−0.5	3	12.3	−0.1	0

ICD = *International Classification of Diseases* (1955 edition).
S.D.D.% = standard deviation of death rates when mean monthly rate is 100.
B = percentage change in death rate per degree Centigrade.
VAT% = percentage of total variance associated with temperature.
Standard deviation of monthly temperatures = 5.02°C.
Significance of VAT estimates: 7.8% = 0.05, 13.1% = 0.01.

tion and strokes. The effect of grouping days together (groups of days axis of the figures) is to reduce the coefficients of variation but this is to be expected as random fluctuations are reduced by the increases in numbers of deaths in each group. This effect is particularly seen in the younger age groups where the numbers of deaths per day are small.

In the left half of the figures is shown the percentage of the variance associated with temperature and in the right the significance of this association. Note the following similarities between the diseases:

1. The association of death rates with temperature is stronger in the old than in the young.
2. The percentage of variance associated with temperature is small and/or insignificant when only one or two days' temperatures are considered but rises most

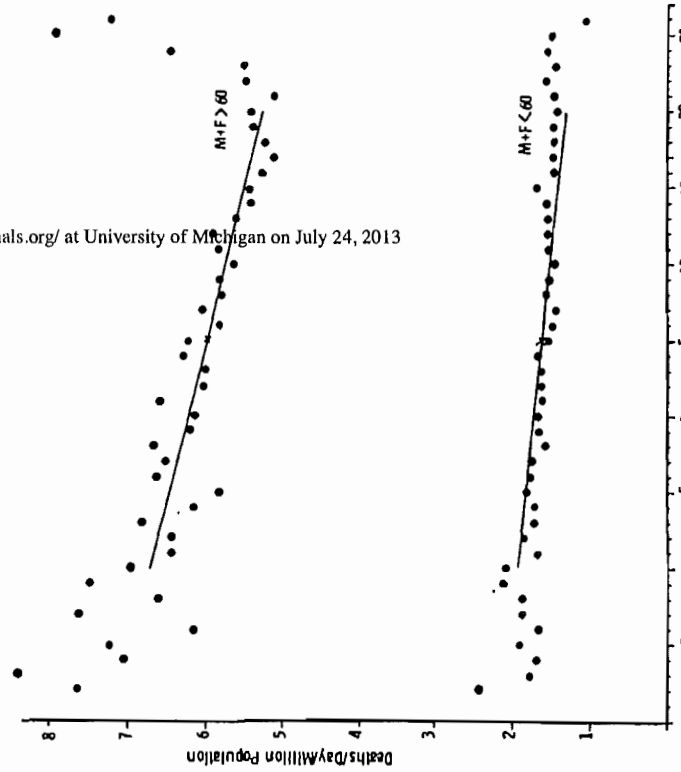

Fig. 2. Analysis of daily deaths from myocardial infarction in New York (1965–8).

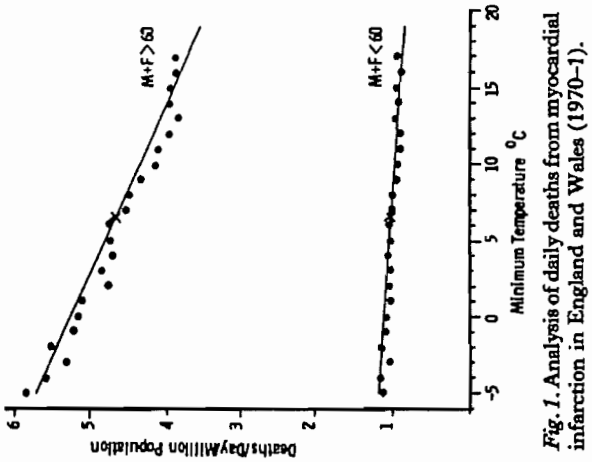

Fig. 1. Analysis of daily deaths from myocardial infarction in England and Wales (1970–1).

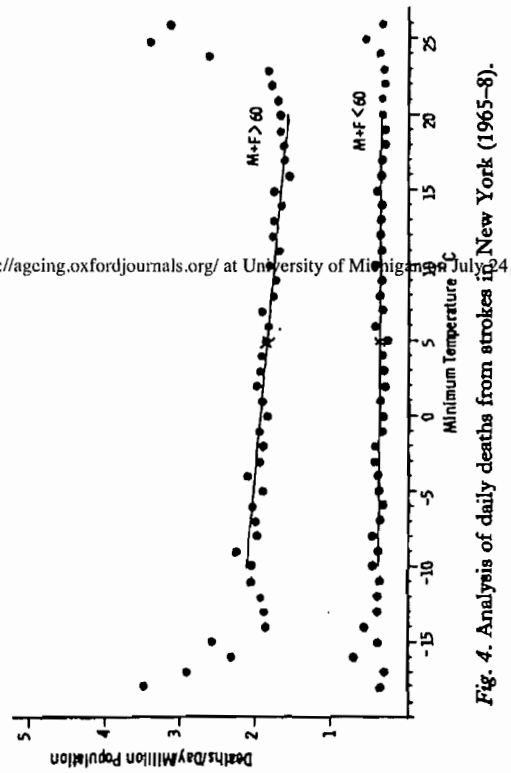

Fig. 4. Analysis of daily deaths from strokes in New York (1965–8).

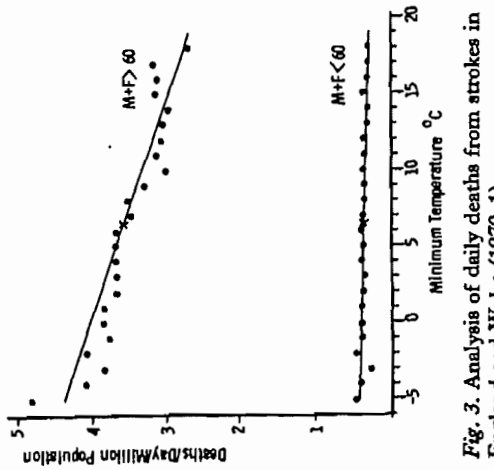

Fig. 3. Analysis of daily deaths from strokes in England and Wales (1970–1).

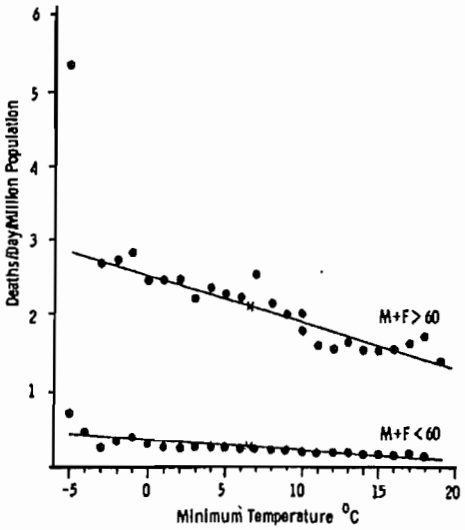

Fig. 5. Analysis of daily deaths from pneumonia in England and Wales (1970–1).

Table II. Relationships between temperature and death rates from myocardial infarction, strokes and pneumonia in England and Wales and New York

Diagnosis	*Age*		*Age specific death rate at mean temp. Deaths/day/M*	*Slope of regression as percentage of mean death rate with temp. ranges*		
				−10°C	*−10 to +20°C*	*+20°C*
Myocardial infarction	<60	E&W	1.27		−1.16	
		NY	1.93	−0.97	−0.75	+0.46
	60+	E&W	24.51		−1.87	
		NY	35.49	−1.43	−0.79	+8.65
Strokes	<60	E&W	0.43		−1.34	
		NY	0.43	−1.44	−0.51	+2.77
	60+	E&W	18.87		−1.97	
		NY	10.97	−7.89	−0.89	+15.14
Pneumonia	<60	E&W	0.34		−5.38	
	60+	E&W	2.89		−4.66	

steeply along the 'group of days' axis to a peak at about 7–10 days. There is then a tendency for a slight fall followed by a further slower rise. From this we can deduce that short-term temperature changes (less than about two or three days) are associated with little change in death rates. However there appears to be a medium-term effect peaking at about a week and then a more sustained, long-term effect.

Note also the following differences between the diseases. In the case of myocardial infarction the association of death rates is strongest with temperature 1–2 days

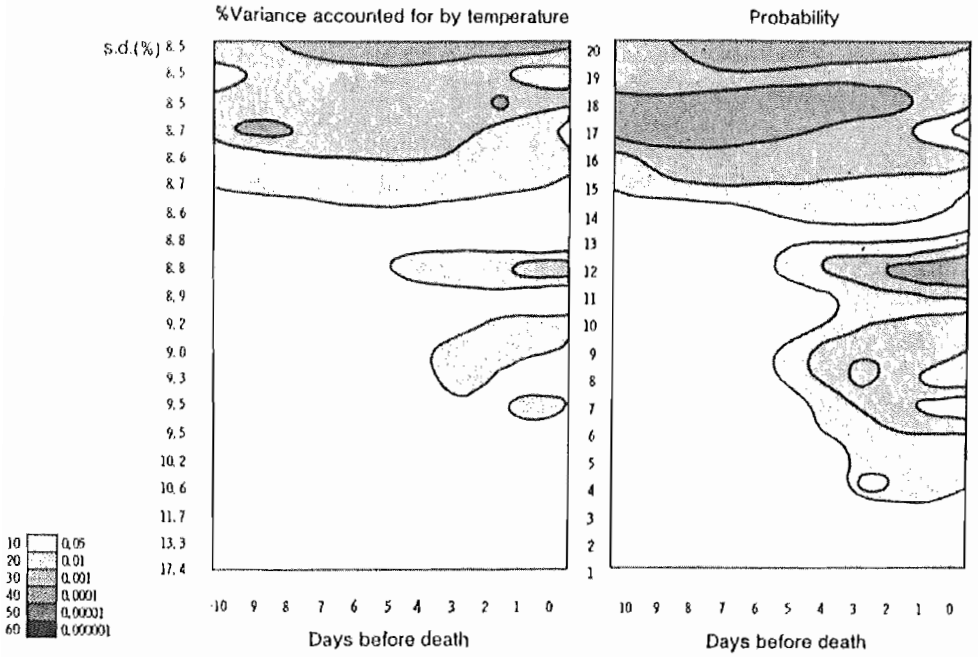

Fig. 6. Deaths from myocardial infarction in those below 60 years in England and Wales (1970–1).

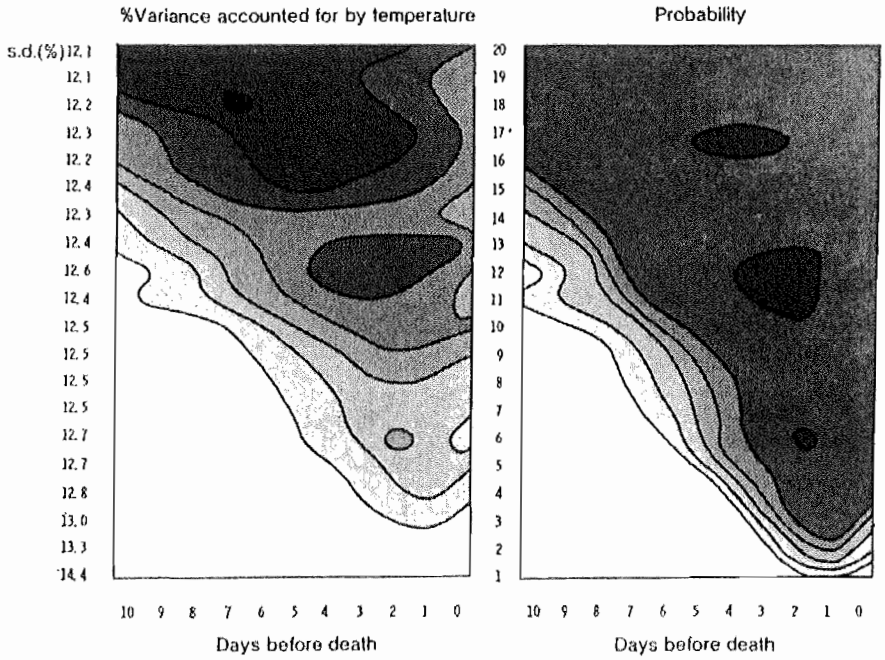

Fig. 7. Deaths from myocardial infarction in those aged 60 years and over in England and Wales (1970–1).

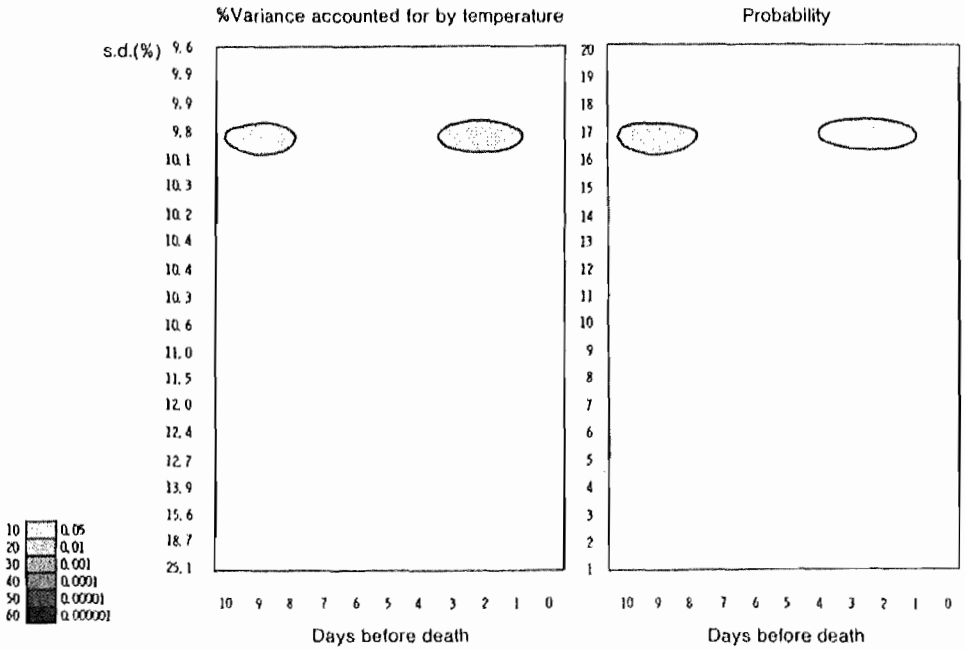

Fig. 8. Deaths from strokes in those below 60 years in England and Wales (1970–1).

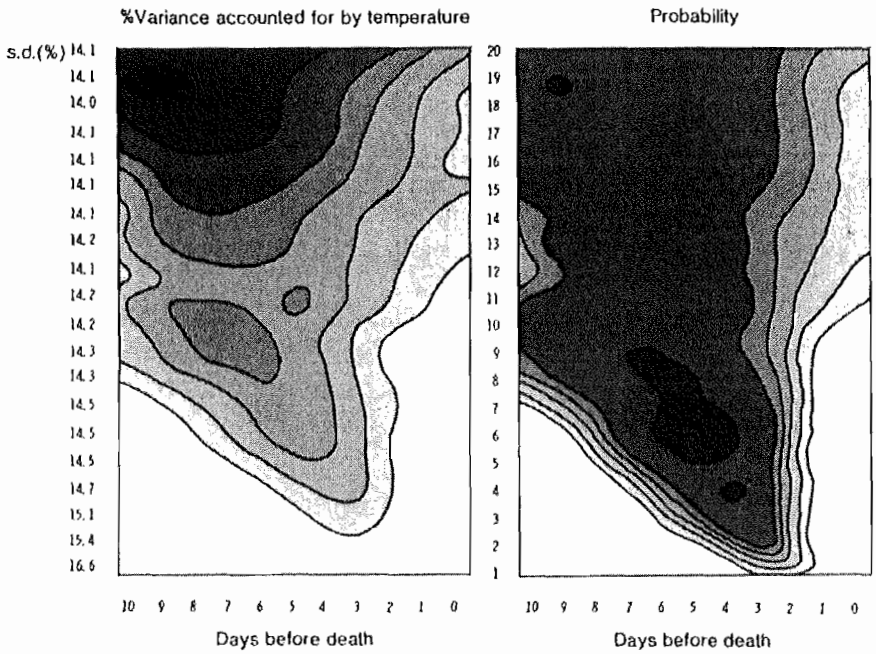

Fig. 9. **Deaths from strokes in those aged 60 years and over in England and Wales (1970–1).**

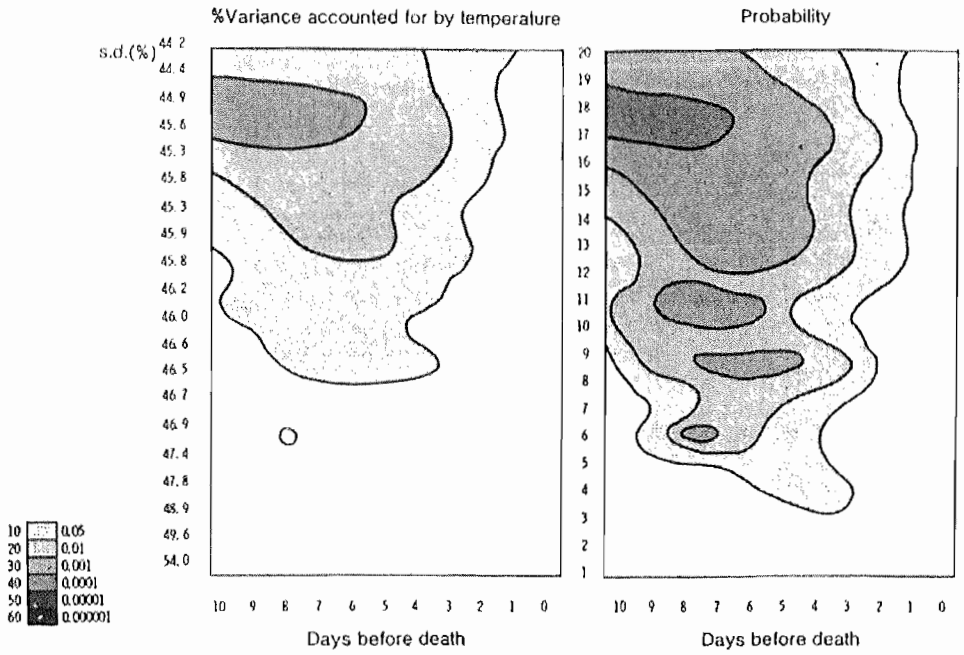

Fig. 10. Deaths from pneumonia in those below 60 years in England and Wales (1970–1).

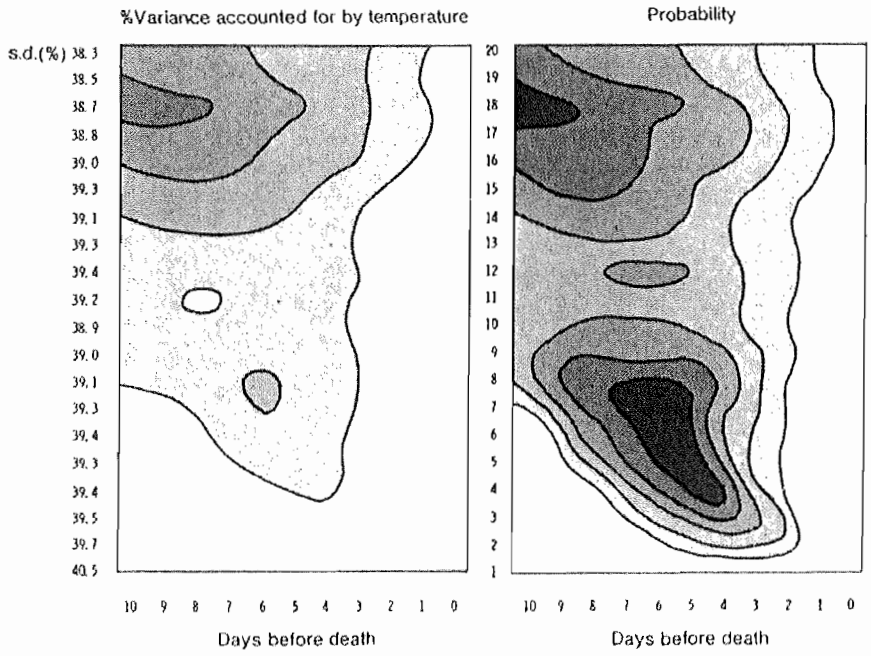

Fig. 11. Deaths from pneumonia in those aged 60 years and over in England and Wales (1970–1).

before death, in strokes 3–4 days before death and in pneumonia 4–6 days before death. In addition, the increase in accounted-for variance is very steep in the older subjects in the case of myocardial infarcts and strokes and much less steep in the case of pneumonia.

The effect of duration of temperature change ('runs of days effect')

The results are shown in Table III. Note that the length of time the temperature remains in a given range affects the death rates differently when the weather is hot or cold. At the lower end of the temperature range the regression of deaths on duration of the 'run of days' is positive, i.e. there are more deaths the longer the 'run of days', while at the higher end of the temperature range the reverse is the case. The longer the 'run' the fewer the deaths.

Table III. Relationship of temperature and duration of temperature change on death rates from myocardial infarct, strokes and pneumonia (1970 and 1971) in England and Wales

		M+F <60 years		*M+F >60 years*	
Diagnosis	*Temp. range °C*	*Deaths/ day average*	*Percentage change in deaths/day/day temp in range*	*Deaths/ day average*	*Percentage change in deaths/day/day temp. in range*
Myocardial infarction	<0.1	54.3	1.95	261.5	3.02**
	0.1–4.0	51.7	0.82	239.3	0.05
	4.1–8.0	50.6	0.00	227.0	0.91
	8.1–12.0	47.0	−0.02	204.6	−0.50
	>12.0	44.8	−0.08	191.7	−1.15*
Strokes	<0.1	18.3	1.24	201.4	3.34*
	0.1–4.0	17.7	0.39	183.0	0.86
	4.1–8.0	17.3	1.19	175.9	1.01
	8.1–12.0	16.0	−0.23	155.3	−0.83
	>12.0	15.4	−0.21	145.6	−0.61
Pneumonia	<0.1	18.5	9.63**	145.9	8.78**
	0.1–4.0	13.4	3.89	116.0	3.24
	4.1–8.0	12.5	4.61*	109.6	3.80*
	8.1–12.0	9.3	−1.40	82.5	−0.83
	>12.0	8.4	−2.2	74.6	−0.85

* $P<0.05$; ** $P<0.01$. These relate to the significance of the values for the correlation coefficients associated with these values.

Discussion

Temperature relationship of death rates in a variety of diseases

From Table I it will be seen that with the exception of asthma in both sexes and diseases of the gall bladder and bile ducts in females, all diseases show a decrease in death rates as the temperature rises. The phenomenon is a general one but the variation in monthly deaths as measured by the standard deviation of deaths as a percentage of total variance associated with temperature (VAT%) differs in different diseases.

The respiratory diseases tend to have high standard deviations and steep negative regressions of deaths on temperature. The atherosclerotic diseases (strokes, ischaemic heart disease, hypertension and diabetes) and most of the diseases of the gastro-intestinal tract have somewhat lower standard deviations and less steep regressions but the percentage of variance associated with temperature tends to be somewhat higher than in the respiratory group.

The group of neoplastic diseases typically have very low standard deviations, even less steep regressions and low percentages of variance associated with temperature. In other words, temperature affects or is associated with death rates very little in this group.

The fact that the respiratory diseases show such a strong association of deaths with temperature is not surprising since transmission of airborne infections tends to be higher in cold weather but the fact that in many other diseases there is an even stronger association of deaths with temperature, as seen by the very high values for VAT%, is not generally appreciated.

It is relevant to consider whether respiratory infections might either initiate events which lead to death from other causes or might provide the coup de grâce to those suffering from other diseases. We have already published evidence and arguments against the first possibility in the case of myocardial infarcts and strokes and some of the arguments are applicable to other diseases (Bull 1973, Bull & Morton 1975*b*). The second possibility remains, for pneumonia is a not infrequent terminal event in many diseases. However, terminal respiratory infections cannot account for more than a small proportion of the variance in deaths in most conditions for the following reason. The neoplastic diseases form a group which should be at least equally susceptible to such infections as other diseases, yet they have low overall standard deviations of monthly death rates and low values for the variances associated with temperature.

It is unfortunate that the small numbers of deaths from certain diseases make it impossible to obtain valid estimates of the effect of temperature on their death rates. For example, it would be of interest to have this information on certain endocrine diseases such as thyrotoxicosis or myxoedema, both of which are known to have disturbances of thermoregulation, for this might at least indicate whether one of the possible steps between temperature change and death was a failure of thermoregulation.

The findings in asthma where there is a positive correlation of deaths with temperature are of interest. Unfortunately, here the result is not statistically significant but in any event, even if it was, the effect could be explained by the fact that allergens tend to be commoner in the summer.

We are therefore left with a few clues to a possible causal relationship between temperature change and deaths but the knowledge that the association is a very general one.

Comparisons between England and Wales and New York

Figs 1–5 show that within the range of minimum daily temperature from about −10°C to +20°C there is a linear or nearly linear relationship between temperature and deaths from myocardial infarction and strokes in both England and Wales and New York, but the regression of deaths on temperature is less steep in New York (see also Table II). Below about −10°C and above +20°C death rates rise steeply, especially in the elderly. This phenomenon has been reported previously by Rogot & Padgett (1976). They also

showed that there were variations in the points of inflexion where steep rises occur in different parts of the United States. In the colder parts the inflexions were towards lower temperatures and in the hotter parts towards higher. This they attributed to either an 'overall physiological adjustment or adjustment in life style to the local climate'. The latter seems more likely for in Toronto, where the control of the indoor micro-environmental temperature is almost uniformly good, there is no significant correlation between outside temperature and death rates from these conditions (Anderson & Le Riche 1970). Central heating is less common in Britain than in North America and this may also account for the steeper regressions of deaths on temperature in the middle temperature range in Britain than in New York. The steep upward inflections in the figures for deaths below −10°C and above +20°C, especially in the elderly, are probably due to a combination of factors—the one a failure of physiological adaptation to the extremes and the other a failure of the microenvironmental control systems in the extreme ranges of temperature.

In the case of pneumonia, only data from England and Wales were available. Here the regression of deaths on temperature is steeper than in the case of the other two diseases (Table II and Fig. 5) and unlike them the younger subjects have steeper negative values for the regression. This may be due to the fact that younger subjects are more exposed to external temperature than older ones.

Age, groups of days and temperature phase shift effects

Several important facts emerge from a study of Figs 6–11 and Table III:

1. The elderly show a much stronger association between temperature and death rates than younger subjects. It is extremely unlikely that this could be due to greater exposure of elderly subjects to the external environment and therefore probable that they fail to adapt physiologically to temperature change. Indeed it is known that this is the case. From various surveys in Britain since 1966, it has been shown that with increasing age there is a progressive reduction in the ability to sense and control temperature. For example, the elderly are slower than the young to detect temperature differences, to shiver and to sweat (Watts 1972, Fennell & Moore 1973, Cowburn & Fox 1974, Collins et al. 1977). They are less successful in conserving body heat and at low ambient temperatures have body temperatures which are both lower in the mean and wider in range than younger subjects (Fox et al. 1973).

These facts all point to a failure of autonomic control in the elderly and if there is a causal relationship between temperature change and deaths, this failure is likely to be a proximal link in the chain of events leading to death.

2. The duration of temperature change necessary to be associated with a change in death rate is not a very short one. Indeed there appears to be a peak of association at about 7–10 days of temperature change—a medium-term effect, and in addition a long-term effect (see Figs 6–11).

Purely autonomically mediated effects would be expected to occur more rapidly than these results indicate and suggest that some other factor, possibly humoral, is involved. One could postulate that failure of automatic responses in the elderly leads cumulatively to a lowering or raising of the body temperature depending on whether the ambient temperature is high or low. This move from an optimal body temperature might affect the functioning of some critical enzyme system or systems.

3. Comparison of Figs 6–11 shows significant differences between the three diseases in respect of interval between temperature change and death.

In the case of myocardial infarction the temperature 1–2 days before death is more relevant than the temperature on the day of death or on days three or more before death. In an earlier publication (Bull & Morton 1975*a*) we have shown that this phase relationship between temperature and death persists even when allowances are made for the length of time it takes for individuals to die from this condition after onset.

In the case of strokes the temperature 3–4 days before death is more relevant than earlier or later days and although the evidence is weaker in this case than in the case of myocardial infarction (Bull & Morton 1975*a*) it does seem that the interval is unlikely to be simply due to the fact that it takes somewhat longer to die after onset of a stroke than after a myocardial infarct.

In the case of pneumonia the phase shift between temperature and death is even greater (about a week) but here it would be reasonable to postulate that it takes on average some days before the infection proves fatal.

At all events, in the case of both arteriosclerotic diseases there appears to be a significant interval between the temperature change and the onset of the episode leading to death, a fact which is potentially important in that if one could identify a link in the chain of events leading from temperature change to death one might be able to break it and avert the fatal outcome.

Conclusions

In recent years there has been much medical and public interest in hypothermia as a cause of death in the elderly and most publications on the subject have been concerned with the effects of very low temperatures on older people. The present study and those preceding it show that there is a close association between temperature and death rates from *most* diseases and at *all* temperatures.

The studies to this point have not established a clear chain of events leading from a change in external temperature to death. Nevertheless, it remains very likely that changes in external temperature *cause* changes in death rates especially in the elderly. Detailed analysis of the relationship between temperature change and deaths from a few diseases suggests that the relative inefficiency of autonomic control of the elderly is in itself not sufficient to account for the association. It seems more likely that this inefficiency permits a change in body temperature which in turn gives rise to some change which after an interval (which varies somewhat between diseases) leads to death. The change is probably a humoral one and if it could be identified it might be possible to prevent its occurrence or counteract its effects.

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Heat-Related Deaths - United States, 1999-2003
Luber, G E;Sanchez, C A;Conklin, L M
MMWR. Morbidity and Mortality Weekly Report; Jul 28, 2006; 55, 29; ProQuest
pg. 796

bly also MDR HIV. The findings in this report, along with increasing syphilis rates, continuing gonorrhea transmission, and the emergence of lymphogranuloma venereum in HIV-positive MSM, reflects a resurgence of unsafe sex among MSM. This behavior also has been associated with increasing use of methamphetamine (*7*).

The genotype data collected by NYCDOH indicated a low prevalence of MDR genotypes among persons who had not been treated with ARVs and who had HIV infections diagnosed during June 1, 2004–June 30, 2005. Drug-resistant HIV compromises the effectiveness of standard ARV regimens and can limit the treatment options available to persons with newly diagnosed HIV infection (*6*). Therefore, CDC has provided funding to four city and 17 state health departments to conduct drug-resistance surveillance on remnant sera obtained from all patients with newly diagnosed HIV infection (*8*). Provisional data from these areas indicate that as many as 15% of these patients are infected with an HIV strain that has mutations associated with resistance to ARVs, and 3.2% have mutations associated with resistance to two or more classes of such medications.[§]

Case reports such as the one described here and results from surveillance of newly diagnosed, drug-resistant HIV infections contributed to recent changes in HIV-1 treatment guidelines issued by the U.S. Department of Health and Human Services (*9*). These guidelines now recommend performing drug-resistance testing before initiation of therapy in patients who have never received ARV treatment. To reduce HIV-associated morbidity and mortality in the United States, public health officials should intensify measures to improve early diagnosis, partner notification, and prevention counseling for persons (particularly MSM) who are HIV positive and should conduct population-based genotype surveillance to monitor the emergence of unusual strains of HIV, particularly those with mutations associated with ARV resistance (*8,10*).

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Heat-Related Deaths — United States, 1999–2003

Heat-related illnesses (e.g., heat cramps, heat exhaustion, heat syncope, or heatstroke) can occur when high ambient temperatures overcome the body's natural ability to dissipate heat (*1*). Older adults, young children, and persons with chronic medical conditions are particularly susceptible to these illnesses and are at high risk for heat-related mortality (*2*). Previous analyses of the risk factors associated with heat-related deaths* have been based on the underlying cause† entered on the death certificate (*4,5*) and have not included decedents for whom hyperthermia was listed as a contributing factor but not the underlying cause of death. This report describes an analysis in which number of heat-related deaths were counted, including deaths in which hyperthermia was listed as a contributing factor on the death certificate. The analysis revealed that including these deaths increased the number of heat-related deaths by 54% and suggested that the number of heat-related deaths is underestimated.

CDC uses information from death certificates categorized by codes from the *International Classification of Diseases* to estimate national mortality trends. These data, collected and submitted by states, were used to determine the number of deaths in the United States during 1999–2003 that had expo-

* Defined as a death in which exposure to high ambient temperatures either caused the death or contributed to it substantially, the decedent had a body temperature at the time of collapse >105°F (>40.6°C), the decedent had a history of exposure to high ambient temperature, and other causes of hyperthermia could reasonably be excluded (*3*).

† The underlying cause of death is defined as the disease or injury that initiated the chain of events that lead directly and inevitably to death. Contributing conditions, or factors, are defined as diseases, injuries, or complications that directly caused the death. A sample death certificate, showing underlying and contributing causes of death, is available at http://www.cdc.gov/nchs/data/dvs/death11-03final-acc.pdf.

sure to excessive natural heat[§] recorded as the underlying cause (code X30 from ICD, tenth revision [ICD-10]), hyperthermia[¶] recorded as a contributing factor (ICD-10 code T67) (*6*), or both.

During 1999–2003, a total of 3,442 deaths resulting from exposure to extreme heat were reported (annual mean: 688). For 2,239 (65%) of these deaths, the underlying cause of death was recorded as exposure to excessive heat; for the remaining 1,203 (35%), hyperthermia was recorded as a contributing factor. Deaths among males accounted for 66% of deaths and outnumbered deaths among females in all age groups (Figure). Of the 3,401 decedents for whom age information was available, 228 (7%) were aged <15 years, 1,810 (53%) were aged 15–64 years, and 1,363 (40%) were aged ≥65 years. The state with the highest average annual hyperthermia-related death rate during 1999–2003 was Arizona (1.7 deaths per 100,000 population), followed by Nevada (0.8) and Missouri (0.6).

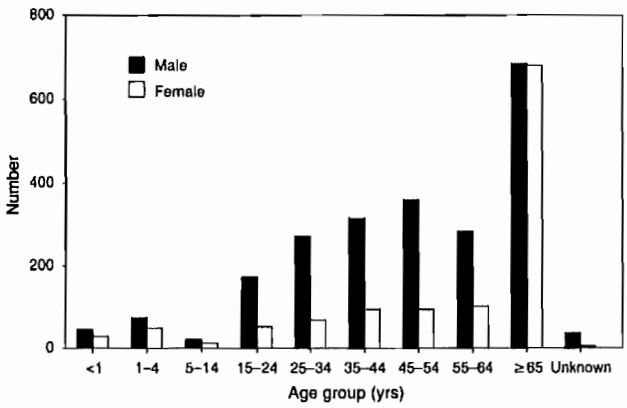
Cardiovascular disease was recorded as the underlying cause of death in 681 (57%) of cases in which hyperthermia was a contributing factor (Table). Approximately 70% of these heat-related cardiovascular deaths occurred among persons with reported chronic ischemic heart disease. External causes (e.g., unintentional poisonings) were documented as the underlying cause of 345 (29%) deaths in which hyperthermia was a contributing factor. Endocrine, nutritional, and metabolic disorders (e.g., diabetes mellitus) were the underlying causes in 38 (3%) of total deaths. All other underlying causes, including infection and psychiatric disorders, accounted for 139 (11%) deaths.

TABLE. Selected underlying causes of death with hyperthermia* as a contributing factor† — United States, 1999–2003

Underlying cause of death	No.	(%)
Cardiovascular diseases	**681**	**(56.6)**
Chronic ischemic heart disease	473	(39.3)
Acute ischemic heart disease	63	(5.2)
Hypertensive heart disease without congestive heart failure	60	(5.0)
Other cardiovascular diseases	85	(7.1)
External causes of morbidity and mortality	**345**	**(28.7)**
Accidental poisoning by and exposure to noxious substances	51	(4.2)
Assault	63	(5.2)
Other external causes of morbidity and mortality	231	(19.2)
Diseases of the respiratory system	**37**	**(3.1)**
Chronic obstructive pulmonary disease, unspecified	27	(2.2)
Other diseases of the respiratory system	10	(0.8)
Endocrine, nutritional, and metabolic disorders	**38**	**(3.2)**
Unspecified diabetes mellitus	26	(2.2)
Other endocrine, nutritional, and metabolic disorders	12	(1.0)
Mental and behavioral disorders	**29**	**(2.4)**
Mental and behavioral disorders due to alcoholism	21	(1.7)
Other mental and behavioral disorders	8	(0.7)
Diseases of the digestive system	**22**	**(1.8)**
Fibrosis and cirrhosis of the liver	15	(1.2)
Other diseases of the digestive system	7	(0.6)
Other diseases of the nervous, infectious, immune, and genitourinary systems and neoplasms	**51**	**(4.2)**

* Abnormally high body temperature caused by the body's inability to dissipate heat.
† N = 1,203.

FIGURE. Number of heat-related deaths,* by sex and age group — United States, 1999–2003



* Exposure to extreme heat is reported as the underlying cause of or a contributing factor to death (N = 3,442).

Reported by: *GE Luber, PhD, CA Sanchez, MD, Div of Environmental Hazards and Health Effects, National Center for Environmental Health/Agency for Toxic Substances and Disease Registry (proposed); LM Conklin, MD, EIS Officer, CDC.*

Editorial Note: In this analysis, the inclusion of hyperthermia as a contributing cause of death increased by 54% the total number of heat-related deaths during 1999–2003 that would have been counted through inclusion of a heat-related underlying cause alone. Because heat-related illnesses can exacerbate existing medical conditions and death from heat exposure can be preceded by various symptoms, heat-related deaths can be difficult to identify when illness onset or death is not witnessed by a clinician. In addition, the criteria used to determine heat-related causes of death vary among states. This can lead to underreporting heat-related deaths or to reporting heat as a factor contributing to death rather than the underlying cause (*3*). The demographics (e.g., sex, age group, and state) of the decedents described in this report are

§ Heat-related deaths can also be caused by exposure to excessive heat of man-made origin (e.g., from saunas or furnace malfunctions; *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision* [ICD-10] code W92) and can include homicides and suicides involving exposure to excessive heat. Deaths from these causes were not included in this analysis.
¶ Abnormally high body temperature caused by the body's inability to dissipate heat.

consistent with previous descriptions of persons at risk for heat-related deaths (*4,5*).

This analysis also provides additional information on the underlying causes of death in which hyperthermia was a contributing factor. Although this report might still underestimate the extent of overall heat-related morbidity and mortality, the inclusion of hyperthermia as a contributing factor to death provides a more comprehensive view of the actual effects of heat-related illnesses. The association between cardiovascular disease and heat-related death is well established (*7*); this analysis suggests the need for additional investigations of the association between noncardiovascular conditions, such as endocrine and respiratory diseases, and the risk for heat-related death.

Continued exposure to excessive heat can lead to hyperthermia or death. Of the heat-related illnesses, heat exhaustion and heatstroke are the most serious. Heat exhaustion is characterized by muscle cramps, fatigue, headache, nausea or vomiting, and dizziness or fainting. The skin is often cool and moist, indicating that the body's mechanism for cooling itself (i.e., sweating) is still functioning. The pulse rate is typically fast and weak, and breathing is rapid and shallow. If untreated, heat exhaustion can progress to heatstroke (*1*). Heatstroke is a serious, life-threatening condition characterized by a high body temperature (>103°F [>39.4°C]); red, hot, and dry skin (no sweating); rapid, strong pulse; throbbing headache; dizziness; nausea; confusion; and unconsciousness. Symptoms can progress to encephalopathy, liver and kidney failure, coagulopathy, and multiple organ system dysfunction (*2*). Prompt treatment of heat-related illnesses with aggressive fluid replacement and cooling of core body temperature is critical to reducing morbidity and mortality (*2*).

Many heat-related deaths, regardless of whether they are associated with chronic medical conditions, are preventable. During periods of extreme heat, heat-related illnesses can be prevented by avoiding strenuous outdoor activities, drinking adequate amounts of fluid, avoiding alcohol consumption, wearing lightweight clothing, and using air-conditioning. Groups at high risk include young children, persons aged >65 years, persons who do strenuous activities outdoors, and persons with chronic (particularly cardiovascular) medical conditions (*8*).

During heat waves, young children, older adults, and chronically ill persons should be checked frequently by relatives, neighbors, and caretakers to evaluate their heat exposure, recognize symptoms of heat-related illness, and take appropriate preventive action. Regardless of the outdoor temperature, parents and other child-care providers should never leave children alone in cars and should ensure that children cannot lock themselves inside enclosed spaces, such as the trunks of automobiles.

Communities can prepare for heat-related illnesses by creating well-defined heat response plans (HRPs) (*9*). Both governmental and nongovernmental organizations, each with specific roles and responsibilities, can be involved in this planning. HRP protocols and communication tools should be reviewed annually, before the summer months begin. The HRPs should identify populations at high risk for heat-related illness and death and determine which strategies will be used to reach them during heat emergencies. The HRP should also include specific criteria for activation and deactivation of the plan. Postemergency evaluations of HRPs are necessary to make appropriate revisions and improve plan effectiveness.

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Chagas Disease After Organ Transplantation — Los Angeles, California, 2006

Chagas disease is an infection caused by the parasite *Trypanosoma cruzi*. Reduviids (i.e., "kissing bugs") transmit the parasite through infected feces. *T. cruzi* also can be transmitted congenitally and through blood transfusion or organ transplantation. The infection is lifelong if left untreated; the majority of infected persons are asymptomatic, and their disease remains undiagnosed. Although routine serologic testing of organ and blood donors is performed in areas of Latin

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Patterns of Urban Heat-Wave Deaths and Implications for Prevention: Data from New York and St. Louis During July, 1966

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Patterns of urban heat wave deaths in New York and St. Louis during July 1966 indicate not only the impact of environmental stress on heat-susceptible segments of the population but provide clues to the possible prevention of such deaths. While mortality from all causes increased by 36% in New York and by 56% in St. Louis certain subgroups were at substantially higher risk (persons over age 65; census-tract residents with low income, crowding, or poor housing; those with hypertensive, arteriosclerotic, cardiovascular, or other circulatory disease, diabetes, or chronic respiratory disease). The oppressiveness of heat waves in cities is emphasized by increased rates of homicide and by clashes with police in the streets. On the brighter side, pediatric deaths were controlled during heat episodes in both cities, suggesting that medical, social, and environmental measures can intervene.

Unless the general approach to urban heat waves becomes prospective rather than retrospective, one can anticipate episodes of excess mortality during the summers of the 1970's with a human cost of about 136 excess deaths per million per week of heat stress.

INTRODUCTION

The summer of 1966 proved that heat waves (episodes of sustained high temperatures with or without high humidity) in U. S. cities are not a thing of the past, but a recurrent meteorological fact. That significant numbers of persons died during the heat wave in many U. S. cities indicates our failure to develop effective preventive measures for some heat-susceptible segments of our urban populations, and our failure to anticipate the consequences of heat stress in making our cities more livable and more healthful.

METHOD OF EXCESS MORTALITY

The method of excess mortality was developed by epidemiologists during the twenties to assess the impact of epidemics of influenza on populations and to derive ongoing expected curves of mortality which would give the health officer a baseline on which to plot weekly reported deaths (Serfling, 1963). The resulting curve of expected and observed deaths with an estimated tolerance zone for random fluctuation is illustrated for New York for the period April 1966–April 1967 in Fig. 1. Here we see expected weekly death rates plotted as a 5-year, 5-week moving mean with a 95% confidence zone (Weiner, 1968). Superimposed on the expected baseline (which has a summer depression and a winter eleva-

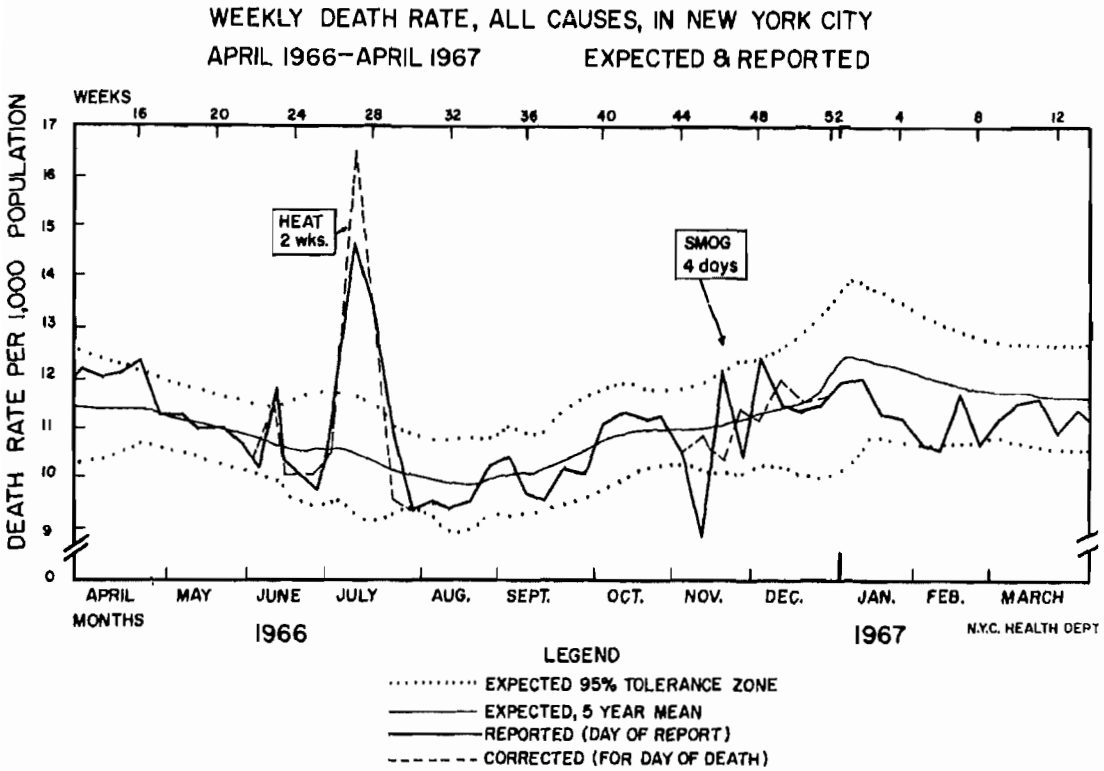

FIG. 1. Fluctuations in death rate in New York associated with episodes of heat (July 2–15) and smog (November 23–26) in 1966, illustrating the method of excess mortality. (Source of data: New York City Health Department.)

tion) are the weekly death rates for the current year, plotted as they are reported by day of report (heavy solid line) and replotted as the rate is corrected for actual day of death (interrupted line). Thus, in New York during one 12-month period there were four periods when the death rate seemed to exceed expectation: mid-April, early June, July, and November of 1966. The largest rise was accompanied by 2 weeks of excessive heat described by the U. S. Weather Bureau as "the result of a merger of three extraordinary high pressure systems that cover the central part of the nation, the Eastern Pacific and the Central Atlantic areas—a coincidence that amounts to a meteorological conspiracy to maintain unrelenting hot weather here. . . ."[1] Note that the peak of mortality is adjusted from about 15 to about 17 per 1000 population after the health department's statisticians corrected for day of death. The small April rise and June rise are brief, not sustained, and not easily explained. The November curve illustrates a well-known episode of 4 days of air pollution which occurred during the Thanksgiving holiday in New York. Although a rise in deaths was widely feared during the hectic days of the episode and although morbidity for chronic respiratory disease cases was reported, the corrected curve of weekly deaths by day

[1] *New York Times*, July 14, 1966, Section C-p. 39, "Relentless heat is almost a plot"; "Record hot spell reaches 101 here."

of death (interrupted line) is well within the tolerance zone of chance expectation. This figure confirms the fact that heat episodes (like epidemic influenza) exert short-term, intense effects on mortality curves which most air pollution episodes do not.

INTERURBAN COMPARISON OF HEAT-WAVE DEATHS

Since Mary Gover's classic article on excess deaths in major U. S. cities during the thirties, there have been few reports comparing the impact of similar episodes in different urban settings (Gover, 1938). Similarities in patterns of deaths should offer some sense of replication and consistency; differences in the patterns should provoke searching questions.

Thus, from a single summer, in 1966, which contained meteorologically similar heat episodes over a wide region, including cities of various sizes, it should be possible to learn more of the effects or lack of effects on urban populations. For simple quantitative comparisons such as numbers of excess deaths by age and by standard cause of death, the computations can be done retrospectively. As a minimum, such interurban comparisons should provide a means of grouping cities by severity of heat-associated mortality. For more complex analysis of heat wave effects one must focus on two or more cities in greater detail with proportionately greater effort.

DEFINING A HEAT WAVE

Defining a "heat wave" requires that some description and interrelation be made between the meteorological features and the mortality features of the episode. The more precise the definition of the heat episode is in terms of days of record heat, or of comfort-index days above a certain level, or of rises of mortality above a certain percentage, the more consistent is the methodology. On the other hand, we are only on the threshold of measurement in the sense that we do not know as yet how many times in several recent summers has a given city experienced heat episodes without a concomitant rise in mortality; only the noteworthy episodes are recorded or studied. From the viewpoint of prevention, the well-documented "escape" of an urban population from heat-wave conditions should be of equal interest to the "failure" of such a population to cope with heat stress.[2] At the present level of surveillance of heat-related deaths in U. S. cities, there is little risk of overreporting; only occasionally does the National Center for Disease Control note the rise in mortality in early or late summer which affects groups of reporting cities which the epidemiologists are following primarily for signs of epidemic influenza. Given that we have a relatively small sample of heat episodes in large cities for study, what new information can be gained that might be of use in considering recommendations for prevention?

In Table I data are summarized from an in-depth study of mortality rates before and during heat waves which gripped New York and St. Louis during July of 1966. The definition of the heat-episode period is arbitrarily selected to in-

[2] Personal communication: Dr. Thomas Downs, University of Texas School of Public Health, Houston, Texas, April, 1971.

TABLE I
COMPARISON OF PATTERNS OF HEAT-WAVE MORTALITY IN NEW YORK AND ST. LOUIS
JULY, 1966

Characteristic	New York	St. Louis
Population at risk, approximately	7.8 million	728,000
Duration of heat wave	14 days	28 days
No. days over 90°F	12	24
Mortality		
Excess deaths—estimated number[a]	1181	618
All ages—proportion rise	36.3%	55.8%
65+ years—proportion rise	52.6%	81.1%
"Rate" per million per week	75.7	197.1
Race		
White	39%	41%
Nonwhite	20%	119%
Sex		
Male	25.3%	28.0%
Female	50.4%	57.5%
Race—sex group at highest risk[b]	WF 56.2%	NWF 140.1% NWM 88.6%
Range of excess deaths by residence (census tract)	10% to 140%	−18% to 260%

[a] Applying the method of excess mortality to an arbitrary control period (see text): For New York—2 weeks of "average" mortality during May 7–20, 1966; for St. Louis—4 weeks of mortality during July of 1965 (7/2–7/29).

[b] WF = white female, NWF = nonwhite female, NWM = nonwhite male.

clude 14 days in New York (July 2–15) and 4 weeks in St. Louis (July 2–29); similarly the choice of a control or comparison period varies. A reasonable choice for New York might have been a 2-week period in June preceding the period in July, such as June 2–15; but we have already noted in Fig. 1 that an abnormal rise in mortality occurred in mid-June for unknown reasons; therefore, a preceding period in May when mortality appeared more "usual" was the logical choice.

The major finding in Table I is that approximately 1181 persons in New York and 618 persons in St. Louis died during the heat episode who would have lived beyond the summer of 1966. One may ask, how many of these deaths may be directly attributed to heat? In St. Louis some 130 of the deaths were assigned to the international category E931 (death due to excessive heat and insolation) but in New York only a handful of deaths were so coded, preference being assigned in New York to underlying circulatory and degenerative conditions. If one accepts the usefulness of the method of excess mortality (as influenza epidemiologists have found over the years) one can overlook differing customs of health departments and practicing physicians in coding deaths and concentrate on the distinct patterns of mortality in the available data.

The impact of the heat wave was greater on the city of St. Louis than New York because deaths were up 55.8% compared to 36.3%. The age group over 65 years was hardest hit by the heat stress, 52.6% rise over expected in New York

and 81.1% in St. Louis.[3] The fact that the cities have different populations at risk and had heat stress of different duration (16 days of maximum temperatures over 90°F in New York compared to 24 such days in St. Louis) may be adjusted by expressing the deaths as a rate per million population per week. The rate for St. Louis clearly exceeds New York, 197.1 compared to 75.7. The combined deaths for the two cities gives a rate of about 136 excess deaths per million per week, or about 19–20 excess deaths per day of a severe urban heat wave. The other features of Table I summarize the data from analysis by sex and race and residence, and indicate clearly that white women were the highest risk group in New York (56.2% rise compared to 36.3% for all persons), that nonwhite women and nonwhite men were higher-risk groups in St. Louis (140.1% and 88.6% rise compared to 55.8% for all persons), and that place of residence of the deceased varied enormously from low areas of 10 to −18% excess mortality to high areas of +140 to +260% excess mortality. What are the reasons for such interurban and intraurban variations in deaths precipitated by urban heat stress? Assuming that differences of these dimensions in such large urban populations are unlikely to occur by chance, one must consider the critical variables in heat-wave survival (based on facts derived from years of clinical, military, and industrial research): duration and severity of heat load; physiologic reserves of the host; environmental or medical methods of arresting the progression of heat illness (Burch and DePasquale, 1962; Leithead and Lind, 1964; Minard *et al.*, 1957; Coburn and Reba, 1966; Lee and Henschel, 1966; Schuman, 1962).

VARIATIONS IN HEAT WAVE MORTALITY BY AGE AND BY CAUSE OF DEATH IN NEW YORK

Clues to the failure to survive the challenge of a heat wave are evident from the data summarized in Table II, and in Figs. 2 and 3. The size of the New York population at risk (7.8 million residents) lends confidence to one's analysis of deaths by subgroups. Thus, in Table II one is reminded that, whereas all causes of death were up by 36.3% over the May control period, only 6 of 16 major causes of death were elevated above expectancy. Arteriosclerotic heart disease accounted for 490 of the 1181 excess deaths and is illustratd in the curve for persons aged 80 years or more in Fig. 2, and in the curve for arteriosclerotic heart disease in Fig. 3. The "time lag" of a day or two before the rise in deaths occurs seen in Fig. 2 is most evident for persons aged 80 or more, but is more delayed for persons aged 45–64 years of age. The greater delay and lesser height of the curve for middle-aged persons suggest the greater resilience to heat challenge for most persons aged 45–64 years; those who succumb at younger ages would suggest a group of premature, possibly preventable deaths.

[3] For purposes of comparison it should be noted that during the 2-week period ending July 16, 1966 deaths from all causes increased 17.4% among 21 reporting cities in five East North Central states (Ohio, Illinois, Indiana, Michigan, Wisconsin) compared to the similar 2-week period of 1965. Deaths for persons aged 65 or older increased by 22.5% in the same region. These data are calculated from *Morbidity and Mortality Weekly Report*, vol. 15, 1966, CDC-PHS-HEW, Atlanta, Georgia 30333.

TABLE II
MAJOR CAUSES OF EXCESS DEATHS IN NEW YORK, JULY 2–15, 1966

Cause of death	Heat weeks (observed)	Control weeks (expected)	Excess in heat wave	Excess as % of expected
All causes	4431	3250	1181	36.3
Homicide	31	13	18	138.5
Diabetes	115	53	62	117.0
Miscellaneous[a]	215	115	100	87.0
Respiratory	350	190	160	84.2
Hypertension	150	99	51	51.5
ASHD	1691	1201	490	40.8
Genitourinary	68	50	18	36.0
Accidents	142	106	36	34.0
CVA	355	279	76	27.2
Cirrhosis	127	105	22	21.0
Digestive	82	68	14	20.6
Cancer	770	657	127	19.3
Other heart dis.	199	169	30	17.8
Infection	22	19	3	15.8
Infancy	91	88	13	14.8
Suicide	23	38	−15	−39.5

[a] Miscellaneous includes all other major causes of death: hematologic disorders; postoperative complications; endocrine diseases (nondiabetic); neurologic disorders; senility; ill-defined conditions.

The method of excess mortality demonstrates its usefulness in showing that while accidental deaths (including motor vehicle fatalities over the July 4th weekend) were above a comparison period in May by 34.0% this excess was not unusual compared to all causes being up 36.3%. However, if we consider two other forms of violent death, homicide and suicide, a striking pattern of reversal appears in Table II and Fig. 3. After a "lag period" of about 1 week, the homicide rate for New York rises sharply while the suicide rate falls below the expected daily average rate. It would appear that the cumulative effects of unremitting heat can raise one's threshold for suicide but lower one's threshold for murder. Perhaps the anecdote recorded during the 10-day heat wave of August 1896 in New York (3000 persons and 2000 horses dying) is relevant at this point: ". . . on the corner of Bleecher and MacDougal streets, police were hurriedly summoned to stop a knife fight which erupted when one man jokingly asked another if the weather was hot enough for him" (Greenberg and Field, 1965).

The effects of oppressive heat on human aggressive behavior are perhaps so well known that further documentation is repetitious. Unfortunately, massive discomfort and disease does not make the news unless it erupts in violence. It is interesting to note that violence on the streets did not erupt in New York with the temperatures in the nineties while it did occur for two nights, July 13–14, 1966, in Chicago's West Side with the temperature only in the eighties. The set and the setting are obviously important where humans, emotions, heat, and crowding are involved. The reporter of the near race riot with scores of police and demonstrators injured from flying bricks, glass, and fire bombs, suggests a

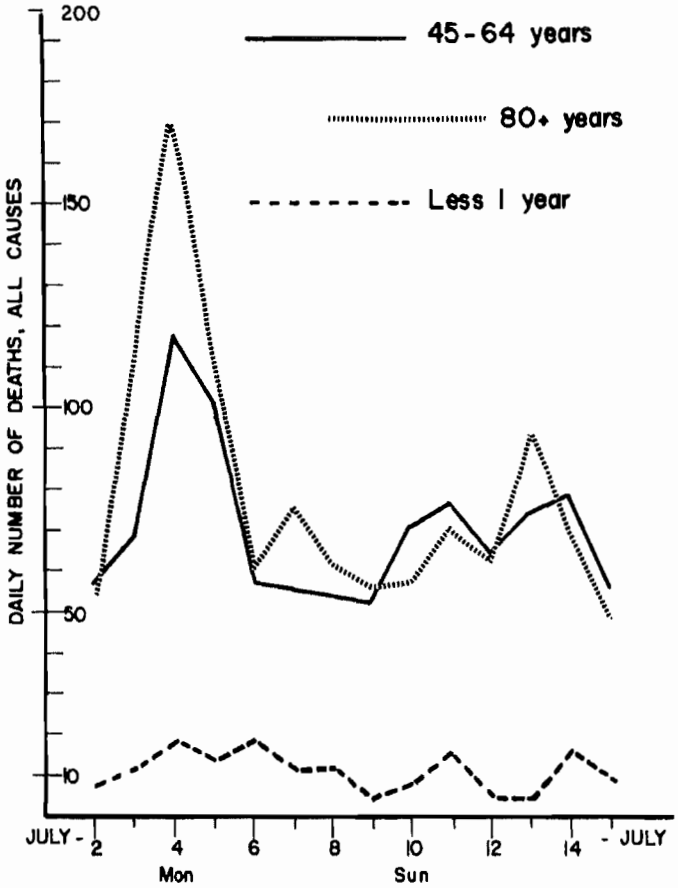

FIG. 2. Daily deaths, all causes, for selected age groups in New York, July 2–15, 1966.

provocative act: "For the second day in a row the police turned off a gushing fire hydrant along Roosevelt Road near the Loop, although it is a summer tradition in Chicago to cool off with gushing hydrants."[4]

Among the causes of death listed for New York the category "respiratory including pneumonia and influenza" showed a rise of 84.2%. This drew the attention of the epidemiologists at the National Communicable Disease Center who wondered about the 2-week rise in this cause of death as the signal for a midsummer (?) outbreak of influenza; strange events sometimes happen, but this possibility proved to be a false alarm. Another possible cause of a rise in deaths from respiratory disease might be the combined effects of stagnant warm air and polluted air in New York. During the episode, the *New York Times* reported, "The pollution index on Wednesday, (July 13) was 13 compared to the usual 6 or 7 for this time of year."[1] The air-pollution levels did not approach the levels of concern that were attained later in New York in November, 1966 of over 20. The additive effects of air pollutants and heat on the cardiopulmonary systems of

[4] Associated Press to Ann Arbor News, July 14, 1966 "Fire bombs, shots fly in wild Chicago melee."

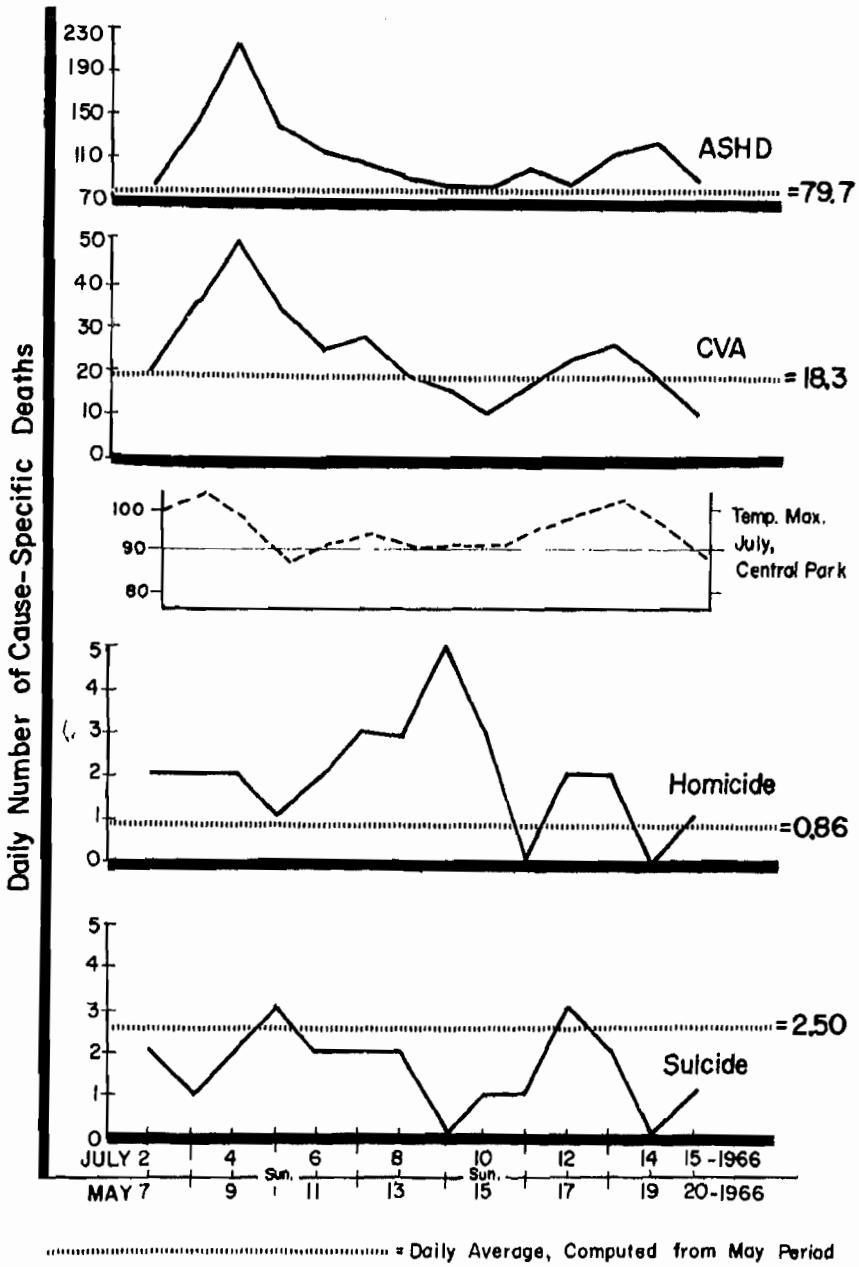

FIG. 3. Daily deaths, selected causes in New York, July 2–15, 1966.

handicapped persons should not be minimized but heat stress would seem to be the dominant factor during July of 1966. A simpler explanation for the rise in respiratory deaths would be the assignment of deaths during the hot spell to the terminal event by many physicians (terminal pneumonia, following stroke, heart failure, chronic cardiopulmonary disease, etc.).

The 34.0% rise in cerebral vascular accidents (CVA) in Table II is not impres-

sive but the timing of the sharp rise for the first week of the heat wave in Fig. 3 coincides with the sharp rise in arteriosclerotic heart disease. It seems that coding of deaths attributable to cerebral strokes in New York differs from coding in St. Louis (see Fig. 5) and tends to understate the problem documented in heat wave studies in Detroit, Los Angeles, and other cities (Schuman *et al.*, 1964; Bridges and Helfand, 1968). It is reasonable to conclude that circulatory handicaps will predispose persons to die during heat episodes regardless of the diagnostic category chosen for certification of death.

Finally, it should be noted from Table II and Fig. 2 that the leading causes of death during infancy (less than 1 year of age) are not elevated during the heat wave. Related categories, such as digestive disorders and infection (diarrhea, dysentery, dehydration), are not increased. These data illustrate the fact that pediatric deaths during urban heat waves are being effectively controlled; while geriatric deaths are not.

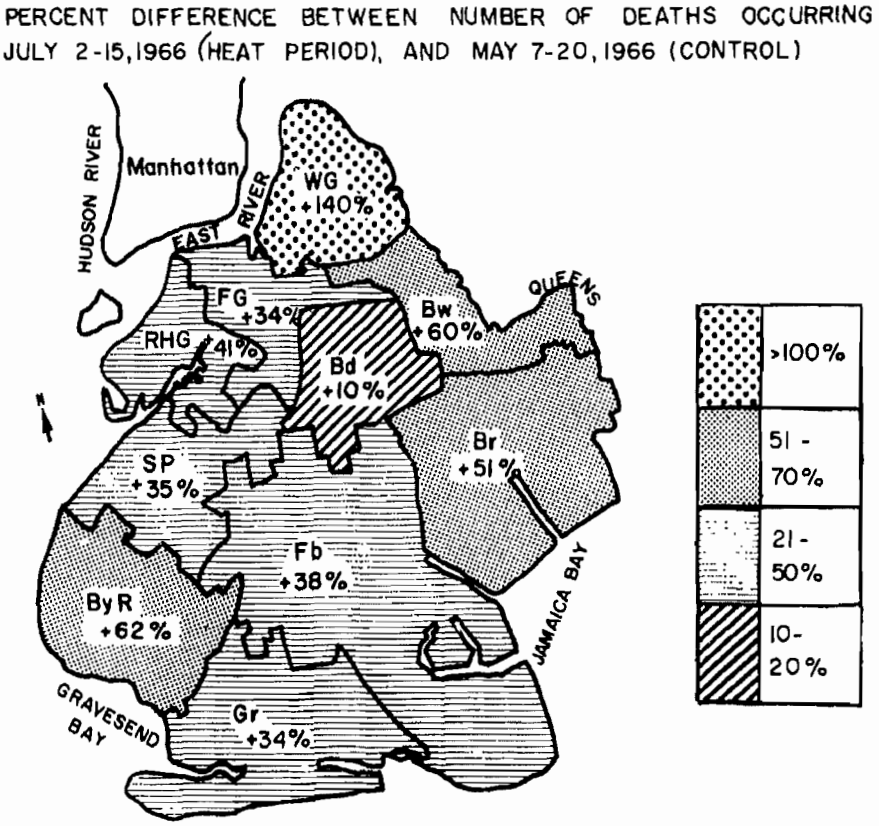

FIG. 4. Distribution of heat-related deaths among the 10 health districts of Brooklyn, July 2–15, 1966.

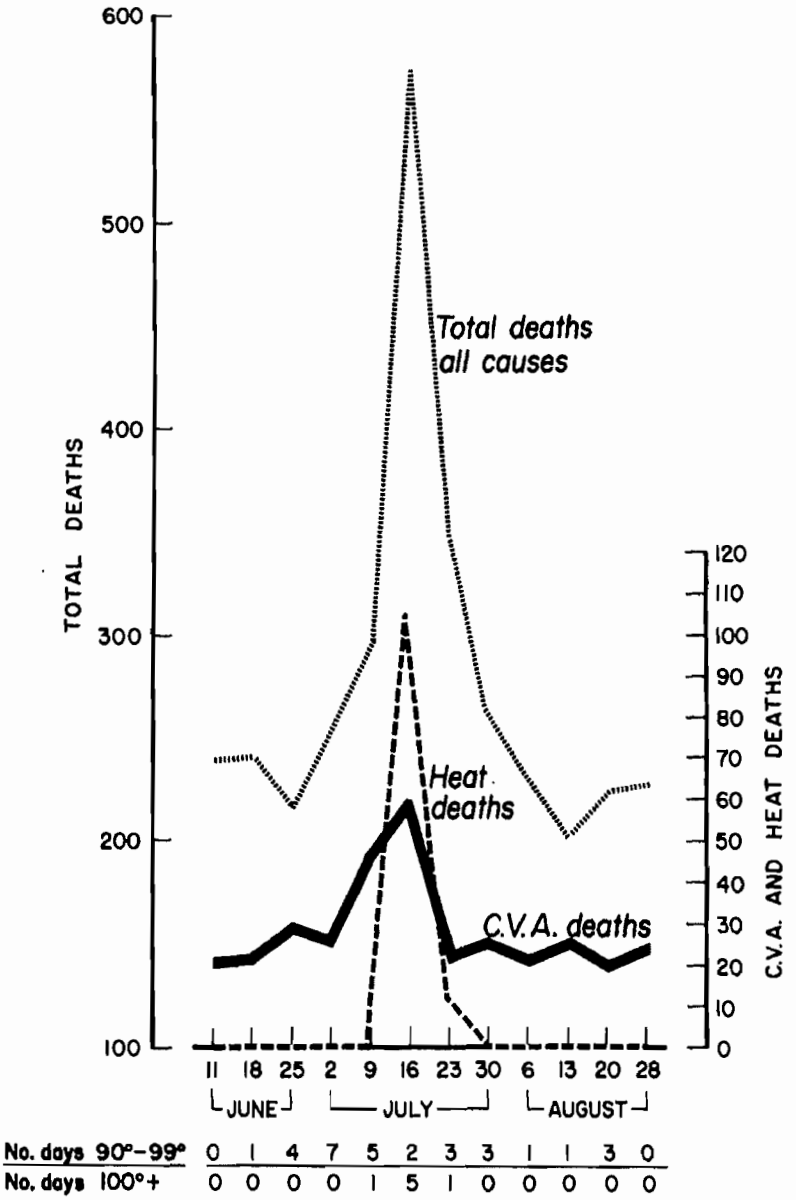

FIG. 5. Weekly temperatures and deaths (all causes and selected causes) in the City of St. Louis, July 1966.

TIMING OF THE HEAT WAVE DEATHS IN ST. LOUIS

Analysis of the heat-related deaths in St. Louis was carried out in similar fashion to that for the data for New York but not in as great detail (cause of death). Most attention was given to the timing of heat-related deaths and their place of residence.

In Fig. 5, the deaths are shown on a weekly scale for June, July, and August in order to emphasize the surveillance possibilities available for earlier recogni-

tion of impending urban heat-wave episodes. In addition to the meteorological forecasting that is available on a probability basis, attention to the weekly patterns of mortality in a city such as St. Louis during the summer months might actually forewarn the community (again on a probability basis) to take greater precautions.

The deaths can be plotted by day of death with more detailed temperature data but the cruder scale serves well enough to emphasize certain essential facts about the timing of heat deaths in St. Louis: (1) Actually 4 weeks in July were characterized by above-normal deaths for all causes; weeks ending July 9, 16, 23, and 30; (2) deaths actually began to rise during the week ending July 2; (3) deaths attributed to cerebral strokes (CVA) began to rise quite sharply and proportionately more than deaths from all causes; (4) heat deaths as such were not coded in St. Louis until 2 weeks after the deaths from all causes began to rise (perhaps the televised daytime all-star baseball game on July 12th with athletes and scores of spectators collapsing focused attention on the heat); (5) the decline in deaths from all causes was slower by 2 weeks than the decline in heat deaths, and (6) the decrements in deaths expected statistically in August to compensate for the rise in July do not become evidence in August for either all causes or for CVA.

In retrospect, it would seem that if attention had been focused on deaths attributable to CVA (or deaths of persons over age 65+ years) as highly susceptible segments of the population during an incipient heat wave, the health facilities and environmental agencies of the community could have been alerted. During the week ending July 2, there were 25 death certificates coded for CVA, well within the 20–30 range of expectation, but by the week ending July 9, there were 47 deaths coded as CVA. On July 5th there were 10 such deaths, followed by eight more CVA deaths on July 6th! Thus, 18 cerebral stroke deaths in 2 days could have aroused suspicion at least 6 days before the baseball game and before general public awareness of a heat wave.

In addition to the possible use of heat-susceptible deaths as a "sentinel" marker of heat-wave deaths, better environmental information should be available to an alert health-protection agency. Thus, the continued use of meteorological data from county-based Lambert Air Field for St. Louis weather is likely to continue a serious underestimate of the actual temperature conditions in the inner city where many of the heat-related deaths occurred. Similarly, the New York temperature readings are from Central Park not the tenements of Brooklyn.

INTRAURBAN VARIATION OF HEAT DEATHS RAISES QUESTIONS

Careful study of heat-related deaths within metropolitan areas by place of residence of the deceased is fraught with methodological difficulties. The findings from geographical analysis of heat deaths in St. Louis and Brooklyn are presented (Tables III and IV, Figs. 4 and 6) in order to raise the epidemiological questions which must be asked, before preventive measures can be evaluated.

Methodologically, one would like to have more accurate census estimates of population at risk for each census tract and demographic characteristics but 1966 was an intercensal period. Nevertheless, it is reasonable to assume that the dis-

TABLE III
DEMOGRAPHIC CHARACTERISTICS OF SELECTED CENSUS TRACTS[a] IN ST. LOUIS, JULY, 1966

Demographic characteristics	High-risk tracts[b]			Low-risk tracts[c]		City of St. Louis
	Ldl	MC	Gfd	NW	Gdv	
Population (thousands, est. 1965)	23.7	15.9	58.6	28.8	18.7	728
Excess deaths						
Number	52	25	57	−8	−3	618
Percent	+260	+179	+110	−18	−10	+55.8
Median age	35.1	26.2	29.6	40.5	43.3	33.6
Race (% nonwhite)	38.9	52.8	97.9	11.1	0.0	35.8
Median family income	$3600	$3400	$3500	$6200	$6700	$5300
Crowding (no. persons/room)	0.70	0.83	0.72	0.55	<0.50	0.64

[a] Census data from 1960.
[b] High-risk tracts are: Ldl = Lindell, MC = Mill Creek, Gfd = Garfield.
[c] Low-risk tracts are: NW = Northwest, Gdv = Gardenville.

crepancy between poor housing districts and better housing districts in Brooklyn or in St. Louis in 1966 did not change too much from the discrepancy as measured in the census of 1960.

When the St. Louis data were first plotted and analyzed (Table III and Fig. 6) geographically, the variation in excess death rates was striking. The rates ranged from −10% and −18% (in Northwest and Gardenville) to +260%, +179%, and +110% (in Lindell, Mill Creek, and Garfield). Could such variation be due to chance? If not by chance, could the environmental stress differ greatly for an area such as Forest Park (+94%) with large acreage of parkland compared to an area of concrete such as Mill Creek (+179%) or Downtown (+141%)? As

TABLE IV
DEMOGRAPHIC CHARACTERISTICS OF SELECTED HEALTH DISTRICTS IN BROOKLYN, JULY 2–15, 1966[a]

Demographic characteristics	Health districts[b]			Brooklyn	NYC
	WG	Gr	Bd		
Population (thousands, 1964)	187	307	281	2615	7840
Excess deaths					
Number	64	42	11	433	1181
Percent	140	33	10	44.5	36.3
Age of residents (% 65 years and older)	10.4	10.1	5.9	9.9	11.2
Income measures					
Median family income	$5500	$6600	$4400	$5100	$5100
Rent (gross median)	$59	$80	$69	$70	$73
Poverty areas[c]	7/10	2/13	10/11	55/115	—
Infant mortality (1964)	31.4	17.5	41.9	29.4	26.8

[a] Census data from 1960.
[b] Health districts are: WG = Williamsburg–Greenpoint, Gr = Gravesend, Bd = Bedford.
[c] Number of census tracts classified as poverty areas based on three indices by Poverty Area Study Office of City Administrator May 1, 1966, New York City.

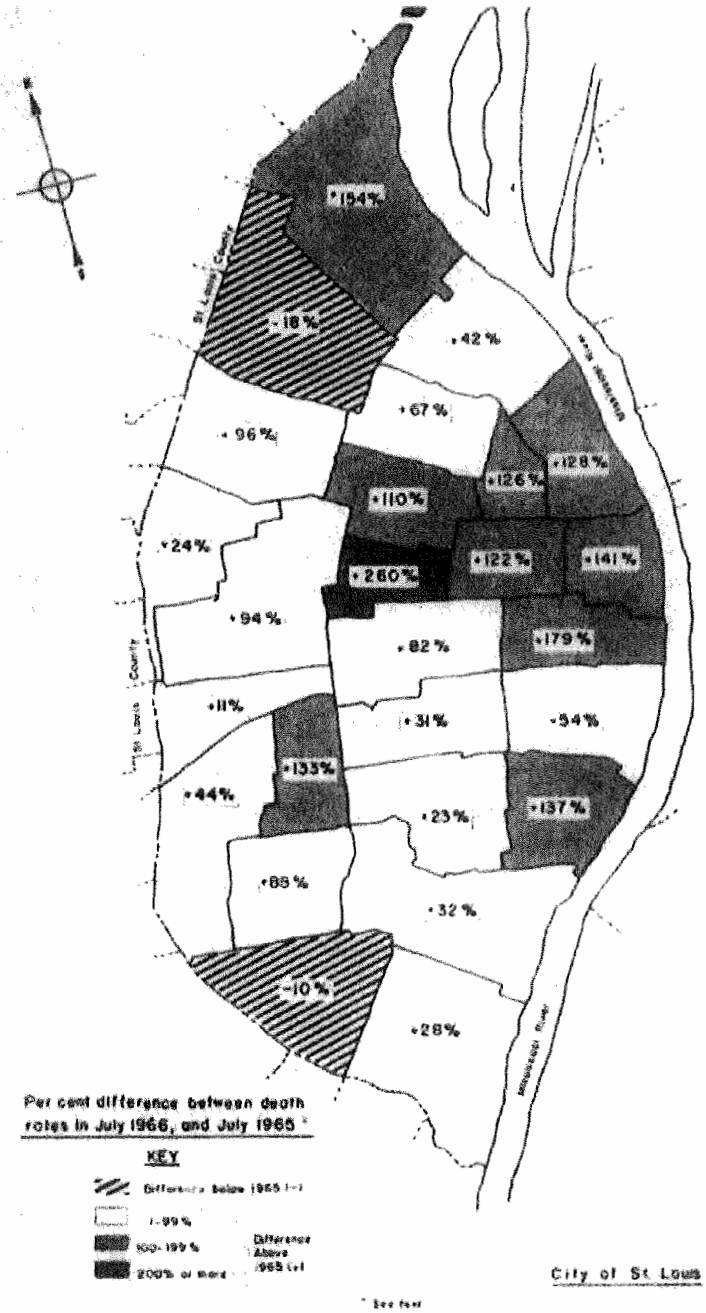

FIG. 6. Distribution of heat-related deaths among the 26 census tracts of St. Louis, July, 1966.

noted previously, the meteorological readings needed to answer such a question are not yet available although isothermal mapping of cities is a well-known technique. Could demographic and housing variables play a part in the variation? The data listed in Table III suggest a pattern, where three selected high-risk

census tracts averaging +183% excess mortality during July 1966 are compared to two low-risk tracts with an average mortality of −14% during the month of record heat. By age composition, it would appear that the residents of low-risk tracts were distinctly older than the St. Louis median age of 33.6 years while Mill Creek and Garfield residents were somewhat younger. The racial comparisons separate the predominantly white low-risk tracts from the high-risk, mixed racial tracts. If there is a mortality gradient by race (percentage black) within the low-risk tracts, the gradient favors being black (+110% for Garfield compared to +260% for Lindell). It seems logical that the last two rows in Table III are most consistent in differentiating high-risk from low-risk census tracts in St. Louis, namely, median family income and crowding (number of persons per room). If we combine these data with those in Table I, we recall that on a city-wide basis, a St. Louis resident was at greatest risk if the resident was over age 65 years, female, and black, and lived in a census tract with low family income and high crowding. Thus, the alleviation of poverty and crowding in the inner city as well as provision of medical and emergency services might save lives dramatically during urban heat waves.

Although the contrasts between tracts are so evident in St. Louis, one could become uneasy about comparisons of census tracts which averaged 28,000 and ranged from 11,000 to 58,000 population. Thus, the opportunity to study a similar heat episode in New York with a population of 7.8 million was considered most advantageous.

INTRAURBAN VARIATIONS IN NEW YORK, JULY 1966

Although the heat-related deaths were up 36.3% for the city of New York (Table I) the five boroughs varied, as follows: Manhattan (20.0%), Bronx (18.8%), Queens (44.5%), Brooklyn (44.5%), and Richmond (37.9%). It was already evident that females were at higher risk than male (50.4% vs 25.3%) that white rates were higher than nonwhite rates (39% vs 20%), and that white females were hardest hit (56.2% vs 36.3%); how would the pattern vary by area of residence? For such detailed study, Brooklyn was selected as one of the two higher risk boroughs; it contains 10 health district serving populations ranging from Red Hook–Gowanus (157,000) to Flatbush (485,000). The excess deaths were plotted geographically as shown in Fig. 4 and again there appeared to be a striking contrast between one low district (Bedford 10%) and one very high district (Williamsburg–Greenpoint 140%). Could these differences be due to chance? With populations of this magnitude at risk, it would seem to be unlikely. Could variations in microclimate between various parts of Brooklyn during the heat wave be important? Such data would be hard to find. Other variables were available for study, shown in Table IV. Another health district (Gravesend) whose rate of 33% is somewhat below the Brooklyn average of 44.5% is included in the table for purposes of comparison. Here we see that the age factor is critical, that the Bedford population is distinctly younger than that of the other districts (5.9% aged 65 or older compared to 10.4% or 10.1%). Somehow despite its poverty (10/11 areas so designated in 1966) this younger population managed to survive the heat wave better than any of the other nine districts in

Brooklyn. Were the high-risk groups of poor infants and children somehow protected during this period of heat stress? If so, the mothers and medical services of the area must deserve some credit. This is in contrast to the high annual infant mortality rate of Bedford (41.9) compared to Brooklyn (29.4). The differences between Williamsburg-Greenpoint and Bedford underscore the importance of age and income: the poor older residents died from heat stress at a greater rate than the poor young residents. Gravesend at the lower range of heat mortality (34%) is characterized by an older population similar to Williamsburg–Greenpoint, but the level of income was distinctly superior for the Gravesend residents. Racial characteristics of the Brooklyn health districts were not investigated. The findings in Table I already suggested a white/nonwhite ratio of 2:1 (39%:20%) for New York in contrast to St. Louis with a ratio of 1:3 (41%:119%). If the race ratios were adjusted for income, housing, and age, would they be similar between the cities? Such analysis would require more detail than the data now permit, but offer a challenge for the next urban study of heat-wave mortality.

DISCUSSION: IMPLICATIONS FOR PREVENTION

The picture of urban heat-wave mortality in the past decade is not reassuring. Given two major cities, such as New York or St. Louis, with a 2-week period of unremitting heat (not necessarily record-breaking), a sharp rise in deaths from all causes is likely to occur. In 1966, the human cost was about 76–197 lives per million residents per week of hot spell. This toll may not seem excessive, but the fact that the rate varied within each city by residential areas and by demographic characteristics suggests that the poor, the disadvantaged, the elderly, and those with circulatory handicaps bore the brunt of the environmental stress. Those with higher income, better medical care, fewer handicapping conditions, air conditioning, or access to the cooler suburbs experienced normal or subnormal death rates during the heat wave period.

On the encouraging side, it should be noted that pediatric deaths due to diarrhea, dehydration, and infections which were prevalent during the summers of the twenties and thirties in the U. S. were not increased during the heat waves in either major city in 1966. It would seem that the challenge of the elderly and the circulatory-handicapped residents of the city could be met with the same vigor, intelligence, and scientific knowledge which is working so well for the very young.[5]

The preventive approaches to adaptation to heat-wave stress must include

[5] The age-specific analysis of excess deaths in the age groups under 28 days, 1–11 months, and 1–14 years for St. Louis shows the following pattern: −12 deaths, +14 deaths, and +7 deaths for July 1965 compared to July 1966. The numerical increase for the latter two age groups is small, but the percentage increase is notable. For New York, the increase over 2 weeks was +21 and +49 deaths for age groups less than 1 year and 1–14 years, numerically small but proportionately significantly increases. The cause-specific analysis for the latter age group shows the increase to be among miscellaneous causes, including metabolic disorders (+23 deaths), accidents, including drowning (+11 deaths), cancer (+9 deaths), and respiratory (+8 deaths). Thus, the traditional causes of infant and pediatric deaths during heat waves are notably absent—gastrointestinal disease (no excess deaths) and infectious disease (no excess deaths).

three disciplines: medical, social, and environmental. From an emergency point of view, medical care is primary but from a preventive or longer-term view, environmental change must be planned. During the immediate hectic days of an incipient heat wave, the emergency rooms and personnel are rapidly overwhelmed and a series of heroic but losing battles are fought. Even the medical skills and teamwork so hard earned during the heat wave are likely to be lost from disuse and personnel changes before the next heat wave occurs. During the less hectic days of planning for urban renewal, architects, engineers, and landscapers can provide a margin of safety for beleaguered residents during heat waves—well-spaced parks and ponds; apartments and homes with cross-ventilation capabilities when the air conditioners break down or the power supply is rationed. The challenge for social change and improvement remains and is clearly indicated by these data to be equally needed by the races, by the crowded and poorly housed wherever they live, in the Midwest or the East. Finally, the morbidity, discomfort, irritations, and mental stress of heat waves should not be underestimated in the future as the quality of life comes to be of greater concern to the well-being of cities and city-dwellers.

SUMMARY

An unusual period of heat stress blanketed St. Louis, the Midwest, and New York in late June and mid-July of 1966. Deaths from all causes increased by 24% in three Atlantic states, 17% in five North Central States, 36% in New York, and 56% in St. Louis. Subgroups of the population were hit harder (white females in New York 56%, nonwhite females 140% in St. Louis). Residents of certain areas of the cities were almost unaffected by heat-wave mortality (—18%, —10%) while in other areas deaths were up by 140 to 260%. Poverty, crowding, poor housing, and age are critical factors. Diabetic deaths were up 117%. ASHD and hypertension were increased by 41 to 52% in New York. Homicides were up remarkably while suicides were down during the hot spell. Sudden heat deaths (DOA's) and heat strokes were coded routinely in St. Louis but rarely by New York physicians. The timing of the deaths and the relation to meteorologic conditions makes such variations in nosology irrelevant when the classic method of excess mortality is applied to the data.

Not only do the cities fail to plan for the prevention of the adverse effects of heat stress on residents, but during the emergency conditions the medical facilities and environmental services are easily overwhelmed, the power shortages, lack of air conditioning, refrigeration, ice, relief areas, or emergency personnel become acute. Factories, labor and management, seem to demonstrate more concern for their workers than hospitals for patients, nursing homes for residents, or cities for their average dwellers.

Surveillance of incipient meteorologic conditions is feasible on a probability basis, and daily monitoring of city deaths, especially the sentinel CVA group of deaths (or persons aged 65 and older) could provide a week's warning before the full brunt of heat-wave deaths occur.

Unfortunately, the grim patterns of urban heat deaths in the decade of the sixties are so consistent between cities (New York, St. Louis, Los Angeles, De-

troit, Madison–St. Clair) that it is predictable that heat-wave episodes will recur in the seventies, unless the problem gains wider attention. Is it possible that the infrequency of heat waves keeps them in the realm of the supernatural rather than the scientific area of our thinking? The possibilities for prevention of heat-wave deaths are limited but they are within the range of our knowledge and skills.

ACKNOWLEDGMENTS

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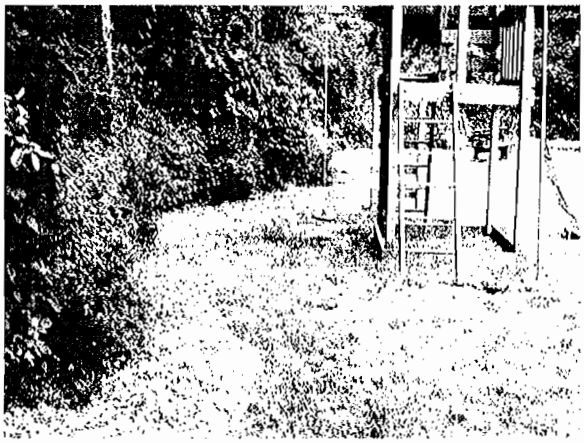
pg. 369

FIGURE 3. Exposure to ticks can be reduced by creating a buffer zone of wood chips or gravel between forest and lawn or recreational areas



Photo/KC Stafford, Connecticut Agricultural Experiment Station

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Impact of Heat Waves on Mortality — Rome, Italy, June–August 2003

During June–August 2003, record high temperatures were reported across Europe; Italy was one of the countries most affected. To assess the impact of the summer 2003 heat waves on mortality, the Rome Local Health Authority analyzed temperature and daily mortality data for June–August 2003. This report summarizes the results of that analysis, which indicated that an estimated 1,094 excess deaths occurred during three major heat wave periods in 2003, an increase of 23% compared with the average annual number of deaths during 1995–2002. Improvements have been made in warning systems and prevention programs that target persons at high risk to reduce excess mortality during future heat waves.

Data on daily deaths during June–August 2003 were obtained from the Mortality Registry Office of Rome. Deaths caused by injury and poisoning (*International Classification of Deaths, Ninth Revision*: 800–999) and deaths that occurred outside Rome were excluded from the analysis. Maximum apparent temperature (MAT)* was defined as an index of human discomfort on the basis of air temperature and dew point temperature (*1*). A major heat wave period was defined as MAT >90th annual percentile and an increase of 4° F (2° C) compared with the previous day. Daily excess mortality was defined as the difference between the number of deaths observed on a given day and the smoothed average daily value for the reference period (1995–2002). Confidence limits were determined by assuming a Poisson distribution. Association between excess mortality and socioeconomic status was evaluated for the census tract of residence by using a deprivation index based on education, occupation, unemployment, number of family members, overcrowding, and household ownership (*2*).

During June–August 2003, the mean daily temperature was 5° F (3° C) above the mean for the reference period, and MAT was 95° F (35° C), compared with 88° F (31° C) for the reference period. During June–August 2003, MAT was >91° F (>33° C) (90th annual percentile) on 55 days (72%), compared with 35% of days during the reference period. Three major heat wave periods occurred during June–August 2003. The first episode (June 9–July 2) registered a mean MAT of 97° F (36° C), with peaks of 100° F (38° C) and 104° F (40° C); the second episode (July 10–30) had a mean MAT of 97° F (36° C) and registered two peaks >104° F (>40° C); and the third episode (August 3–13) was shorter but registered a mean MAT of 100° F (38° C), with 3 days >104° F (>40° C).

* Calculated as $-2.653 + 0.994Ta + 0.0153 (Td^2)$.

TABLE. Number and daily mean of deaths reported and expected and number of estimated excess deaths, by selected characteristics — Rome, Italy, June–August 2003

Characteristic	No. deaths reported		No. deaths expected		Estimated no. excess deaths	Variation*	
	Total	Daily mean	Total	Daily mean		%	(95% CI†)
Age group (yrs)§	5,894	64.1	4,800	52.2	1,094	22.8	(19.7–25.9)
0–64	840	9.1	870	9.5	-30	-3.5	(-10.0–3.1)
65–74	1,150	12.5	1,084	11.8	66	6.1	(0.0–12.2)
75–84	1,919	20.9	1,484	16.1	435	29.3	(23.6–35.1)
≥85	1,985	21.6	1,362	14.8	623	45.7	(39.3–52.1)
Sex§							
Male	2,689	29.2	2,379	25.9	310	13.0	(8.7–17.3)
Female	3,205	34.8	2,421	26.3	784	32.4	(27.8–37.0)
Location of death¶							
In hospital	2,223	36.4	2,088	34.2	135	6.4	(2.0–10.9)
Out of hospital	1,170	19.2	954	15.6	216	22.6	(15.6–29.7)
Socioeconomic level¶**							
High	824	13.5	778	12.8	46	5.9	(-1.3–13.2)
Medium high	1,227	20.1	1,195	19.6	32	2.7	(-3.1–8.4)
Medium low	1,144	18.8	1,016	16.6	128	12.7	(6.1–19.2)
Low	789	12.9	670	11.0	119	17.8	(9.5–26.0)

* Number of excess deaths multiplied by 100, divided by number of deaths expected.
† Confidence interval.
§ Data for June–August 2003.
¶ Data for June–July 2003.
** A factor analysis was used that divided persons on the basis of the 20th, 50th, and 80th percentiles into four socioeconomic categories as shown.

Daily mortality trends followed temperature trends, with peaks in deaths occurring on the same day as or ≤2 days from peaks in MAT. During June–August 2003, both temperatures and mortality trends were consistently above the long-term trend, and total mortality was 22.8% higher than expected, with an estimated 1,094 excess deaths (Table). The first heat wave was associated with an increase in mortality of 352, with peaks in mortality of 87 deaths on June 14 (daily excess mortality: 58%) and 88 deaths on June 26 (daily excess mortality: 54%), corresponding to peaks in MAT of 100° F (38° C) and 104° F (40° C), respectively. During the second heat wave, 319 excess deaths occurred; a peak in mortality (89 deaths) occurred on July 25 (daily excess mortality: 68%), with a lag of 1 day. A second peak in mortality (84 deaths) occurred on July 27 (daily excess mortality: 55%), coinciding with the peak MAT (106° F [41° C]). During the third heat wave, 170 excess deaths were reported. Peaks in mortality occurred on August 8 and August 12, with 77 (excess mortality: 48%) and 78 (excess mortality: 59%) daily deaths registered ≤2 days after peaks in MAT (102° F [39° C] and 106° F [41° C]).

Excess mortality occurred only among persons aged ≥65 years and increased with age, with the greatest impact on persons aged ≥85 years (623 deaths; excess mortality: 45.7%) (Table). The greatest increase in mortality occurred among females (estimated daily excess: 35%), reflecting the higher proportion of women aged ≥85 years (age distribution: women, 72%; men, 28%). A higher excess mortality was observed for out-of-hospital deaths (22.6%) than for in-hospital deaths (6.4%). Excess mortality was associated with socioeconomic status, with an excess mortality of 5.9% among persons in the highest level and 17.8% among those in the lowest level (Table).

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Editorial Note: During summer 2003, the early onset of hot weather, unusually high temperatures, and prolonged heat-stress conditions caused extreme peaks in mortality throughout Europe. The total number of heat-related deaths that occurred during the summer 2003 heat wave is unknown. However, excess mortality data from five countries (France, Italy, Portugal, Spain, and the United Kingdom) indicate that the potential impact of heat waves on health was underestimated and that health authorities were unprepared to cope with this emergency.

During June–August 2003, record excess mortality occurred in Rome during three intense heat waves. The greatest increase in mortality occurred among persons aged ≥65 years living in the most economically disadvantaged areas of the city. The high number of excess deaths in this population might reflect the number of elderly persons of low socioeconomic status who remained in the city during the summer. Other

socioeconomic factors that might have an impact on health include poor housing quality, absence of air conditioning, lack of access to social and health services, and individual behaviors (e.g., alcohol consumption and taking medication). Although the third heat wave was shorter, it was more intense, with higher temperatures. Lower peaks in mortality observed during the third wave might be attributed to a reduction in the susceptible population, as observed in other cities (*3*).

Episodes of heat-related mortality in Rome have been reported in previous summers (*4*). In 1999, Rome was included in a World Meteorological Organization project on cities at high risk for heat-related morbidity and mortality. In 2002, the city implemented a heat health-watch warning system (HHWWS) for the prevention of heat-related deaths during heat waves (*9,10*) and a public health intervention program targeted at persons at high risk (e.g., persons aged ≥65 years and those suffering from chronic disease) during extreme weather conditions (*5,6*). In 2001, of the estimated 2.7 million persons living in Rome, 486,000 (18%) were aged ≥65 years (National Italian Institute of Statistics, unpublished data, 2001), and the mean annual number of deaths recorded was 26,000 (*7,8*).

Rome's HHWWS analyzes meteorological forecast data during May–September to predict oppressive air masses and related excess mortality and issues an alarm when these two conditions are forecast. The alarm is upgraded to an emergency when these conditions persist for >2 consecutive days. During summer 2003, the HHWWS called an alarm on 23 days (25%) and an emergency on 20 days (22%). During heat waves, warning bulletins are posted on a municipal website and disseminated to health authorities. Guidelines for the general population and for patients suffering from specific diseases were developed in collaboration with the Association for General Practitioners. The plan is activated on alarm and emergency days to alert public and private subjects (e.g., clinicians, nursing homes, social centers for the elderly, and hospitals) and to provide information to the public (*3*). A telephone assistance service for elderly persons is available 24 hours a day, 7 days a week. This service provides regular check-in calls, counseling, home delivery of food and medicine, and other services to registered users. A network of social services, volunteers, and street units trained to handle emergencies is activated during the summer months. The Rome Municipality continuously informs elderly persons on the location of social centers and public buildings with air conditioning.

In Italy, as in most of Europe, the increasing proportion of elderly persons and the possible effects of global warming could make the susceptible population more vulnerable to heat waves, leading to increased heat-related mortality. To prepare for possible heat waves in 2004, Rome health authorities have improved the technical capacity of the HHWWS and strengthened municipal prevention programs targeting susceptible populations.

Acknowledgment

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Notice to Readers

Alcohol and Other Drug-Related Birth Defects Awareness Week, May 9–15, 2004

The National Council on Alcoholism and Drug Dependence has designated May 9–15, 2004, as Alcohol and Other Drug-Related Birth Defects Awareness Week. This week is a reminder that alcohol and drug use during pregnancy can be detrimental to a mother and her child. Prenatal alcohol use can result in a spectrum of adverse conditions. One of the most severe outcomes is fetal alcohol syndrome (FAS), which includes facial malformations, growth deficits, and central nervous system problems.

Heat-Related Illness

Current Status of Prevention Efforts

Edwin M. Kilbourne, MD, FACP, FACPM

The centuries-old problem of heat-related illness persists, despite our increasingly detailed and extensive knowledge of effective interventions and groups at risk. Substantial heat waves in the United States may claim thousands of lives. What are the reasons for our apparent inability to deal effectively with this (theoretically) entirely preventable cause of fatal illness?

Agent

Is the agent itself (i.e., environmental heat) more intense? Apparently it may soon become so, and perhaps it already is. It is now widely accepted that the long-discussed global warming trend is a reality, and some authors have expressed concern that the higher average temperatures will involve more heat waves.[1] But the gradual increase in average summer temperatures is not, in itself, expected to produce increases in heat-related mortality. Paradoxically, heat seems to cause fewer health problems in characteristically warm areas than in those with a more variable climate; the temperature level required to increase mortality is actually higher in hotter climates.[2,3] It is not the absolute temperature value, but rather the extent of upward deviation from the usual summer temperature that seems to be the key variable affecting mortality.

Unfortunately, many current models of global climate change predict greater meteorologic variability (i.e., a greater number of such deviations from "normal" temperatures), and it is possible that summer meteorologic patterns currently associated with increased mortality will increase in frequency.[2] An increase in heat-related mortality occurring as the result of global climate change is therefore a distinct possibility. Nevertheless, current models linking weather and mortality lack comprehensive validation, and their parameters cannot be calculated with sufficient reliability to allow precise estimates of the impact of climate change upon the death rate, if indeed there is any impact at all.

The extent to which underlying long-term climate change may have influenced acute episodes of increased summer mortality in the midwestern and northeastern United States in recent years is still unclear. Accordingly, public health investigation of these episodes, including the outbreak in Chicago reported by Naughton et al.[4] in this issue of the *American Journal of Preventive Medicine* and other similar investigations in recent years[5,6] have appropriately focused on the hosts and environment.

Host

The hosts in these episodes show common characteristics. Largely, they are debilitated or chronically ill, elderly or middle-aged adults living in non–air-conditioned apartments in the poorer parts of urban areas. They show unusually high rates of psychiatric disease and alcoholism and frequently take anti-cholinergic or neuroleptic medications.[4–7]

A particularly interesting feature of the study by Naughton et al.[4] is the strong association of social isolation with death from the heat. The investigators used a multiple regression procedure to identify the principal determinants (potential causes) of the health outcome. Two of the three most important variables identified reflect social isolation; these variables were (1) living alone and (2) not leaving home every day. Interestingly, the social isolation variables were stronger predictors of heat-related death than other more intuitively appealing attributes of susceptible hosts, such as having heart disease, depression, or other psychiatric illness. An earlier study of Chicago heat deaths during the 1995 heat wave supports these findings.[5] In that investigation, living alone and lacking access to transportation were associated with heat-related death.

Nevertheless, the precise factors connecting social isolation with demise during the heat remain unclear. It is possible that the companionship of others leads to the prompt recognition of early signs of a heatstroke, cardiac event, or other potentially lethal illness that occurs with increased frequency during the heat. However, it is also possible that risk is somehow intrinsic to the person who chooses to live alone and does not arise from the absence of companionship. Under similarly uncomfortable hot conditions, people who live alone

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may behave differently from those who live with others, and these differences could exacerbate risk. Further work is necessary to develop data to resolve this point, which is one that is clearly relevant to the preventive recommendations of Naughton et al.[4] If absence of companionship is the key factor, then the authors' recommendation for making twice-daily contact with isolated persons may be helpful. However, if some other aspect of a solitary individual's life increases risk, then the recommendation may not have much impact.

Environment

The findings of Naughton et al.[4] on air conditioning and fan use are entirely consistent with prior studies and reinforce them. The lesson is simple: Air conditioning protects against the health dangers posed by environmental heat, but electric fans do not.[4,5,8] Increasing air movement so markedly enhances the physiological impact of low ambient temperatures that a new term was coined to describe this effect: the "wind chill."[9] On the other hand, as ambient temperature rises and the temperature gradient between the body and the surrounding environment diminishes, airflow becomes far less efficient in removing heat.[10] Air conditioning remains effective because its effect is to decrease ambient temperature, which is the primary environmental problem. It also diminishes humidity, which facilitates evaporative heat loss.

The inefficacy of electric fans is nevertheless counterintuitive for many people. Governmental and nongovernmental organizations' heat wave relief efforts continued to include distribution of fans for years after the first good evidence that fans were ineffective. Now, because partial-day air conditioning has been shown to be an effective intervention, providing public access to air-conditioned, designated heat wave shelters or other locations has become the preferred strategy. However, providing enough such locations, along with appropriate transportation for the elderly and those with chronic diseases, has been problematic.

Current Status

Although the current and ultimate contribution of global climate change to heat-related mortality is not yet known, climate change is clearly not the main cause of persistent episodes of heat-related death in U.S. cities. There are a number of criteria for identifying persons at high risk. However, it is not clearly feasible for us to contact and involve all high-risk persons in prevention programs that would be adequate to prevent heat-related illnesses.

The most effective environmental intervention to prevent heat-related deaths is home air conditioning. Although the costs of energy and equipment for air conditioning the homes of the high-risk, elderly poor seem prohibitive, it may now be time to at least consider movement in this direction. There is an implicit dichotomy in the societal value placed on winter heating and summer cooling, despite the potentially life-saving nature of both. In many areas, building codes require that equipment be present to warm a rented dwelling to a specific temperature in the winter. However, similar requirements for air-handling equipment sufficient to cool a dwelling in summer are rare. The continuing occurrence of preventable deaths signals that it is time to begin a discussion of how to better serve the health needs of our fellow citizens who are vulnerable to the heat.

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Are heat stroke patients fluid depleted? Importance of monitoring central venous pressure as a simple guideline for fluid therapy

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During pilgrimage season (Hajj) in Saudi Arabia 34 patients with heat stroke (HS) were centrally cannulated to assess their state of hydration and fluid requirement during cooling period. Central venous pressure (C.V.P.) measurements indicated that most victims of heat stroke had normal C.V.P. on arrival at heat stroke centres and may not be fluid depleted. Twenty-two patients (64.7%) had normal or above normal C.V.P. Twelve patients (35.3%) had zero or below zero C.V.P. Six patients (17.6%) had above 10 cmH_2O (range 10—26 cmH_2O) and could have developed acute congestive heat failure and pulmonary edema if they had been transfused at the standard recommended rate of 3—4 litres of fluid during an average cooling time of 1 h as has been practiced in the heat stroke centres to date [1,2]. This study also showed that heat stroke patients should not be briskly transfused because the heart may be affected by heat stroke per se and an unmonitored challenge by brisk i.v. therapy during cooling (which on its own increases preload on the heart due to peripheral vasoconstriction) can lead to acute overload problems. An average of 1 litre of normal saline or Ringer's lactate (crystalloids) was sufficient to normalize C.V.P. during the cooling period and to restore an optimal state of hydration without predisposing to congestive cardiac failure and pulmonary edema — the potential to develop disastrous adult respiratory distress syndrome and disseminated intravascular coagulopathy.

Temperature — Hyperthermia — Heat stroke — Monitoring — Central venous pressure — Therapy — Crystalloid requirements

INTRODUCTION

The pilgrimage (Hajj) takes place between 8th and 12th Dhulhajj, the 12th lunar month of the Islamic Higira calendar. The Higira calendar is shorter than the Gregorian calendar by 11 days, therefore, the Hajj rotates through the different seasons of the Gregorian calendar every 33 years. When Hajj falls between May to September the pilgrims are exposed to very hot weather (local temperature 38—50°C and humidity

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approx. 50%) resulting in many cases of heat illnesses, e.g. heat cramps, heat exhaustion and heat stroke.

Heat stroke is the least common but is the most severe form of heat illness. It is characterized by hyperpyrexia (core temperature > 40°C) anhydrosis and central nervous system disturbances ranging from confusion to coma, with or without convulsions, [3—5] and it results in considerable mortality [3—7]. Several factors predispose to heat stroke. Most common are high ambient temperature and humidity, and excessive physical exertion in unacclimatized pilgrims. Other contributing factors include old age, obesity, diabetes mellitus, respiratory and cardiac diseases.

Victims of heat stroke (HS) may suffer from severe salt and water losses due to excessive sweating, vomiting, diarrhea, unavailability of water at critical times during performance of rigorous pilgrimage rites which continue for several days and inability to get water in case of very old, weak and frail pilgrims.

To date, standard treatment of heat stroke at the heat stroke centres has included body surface cooling combined with rapid intravenous infusion of 3—4 litres of crystalloids [1,2] at room temperature of 20—25°C which resulted in acute overload problems such as acute congestive heart failure and pulmonary edema perpetuating to adult respiratory distress syndrome and coagulopathy.

The authors witnessed an unprecedented number of heat stroke patients with acute pulmonary edema and congestive cardiac failure during the cooling and post cooling period and realized the risk of unmonitored administration of such large volumes of i.v. fluids within a short span of cooling time of an average 1 h.

Assessment of intravascular volume and compliance of C.V.S. to i.v. fluid challenge can be achieved by invasive monitoring more accurately with swan ganz catheter or with central venous pressure monitoring.

The study was started off with a swan ganz catheter in 13 cases [3] and monitoring of C.V.P. reflected a very good co-relation between PCWP and C.V.P.

We therefore studied this problem by monitoring the C.V.P. as a simple guideline for assessment of state of hydration and compliance of the stressed heart to the challenge of infusion customarily being exercised in the heat stroke victims.

SUBJECTS AND METHODS

Thirty-four patients, 7 females and 27 males having heat stroke with mean temperature of 42.1°C (range 40.7—44°C) with a mean age of 55 years (range 31—80 years) were included in this study.

Once the patient was received at the cooling unit (room temperature 20—25°C) i.v. line set up, blood samples were collected for necessary investigation according to a set protocol from the heat stroke medical management committee. O_2 was administered and a 4-channel temperature recorder (Digitron instrument Ltd.) was connected to four different body sites. Three skin surface and one rectal core temperature were simultaneously displayed to know the temperature differential and fall in core temperature during the cooling process. Body surface cooling was started either with conventional cooling [8] by spraying water at room temperature over the muslin covering the patient and fanning from different angles or MAKKAH AL-MUKKARA-

MAH Body Cooling Unit (MMBCU) [9]. Pulse, blood pressure, respiratory rate, core temperature and three skin temperatures at different parts of the body were recorded every 5 min. The cooling was stopped when the patients core temperature dropped to 39°C.

Central venous line was established either through right subclavian or internal jugular veins of either side in the neck with Vygon C.V.P. catheters by the Seldinger technique (size 14—16) or by a peripheral route in the cubital fossa with drum cartridge "Abbott" size 12. Crystalloids were mainly infused through both i.v. lines in a controlled manner and C.V.P. was measured every 3—5 min either by water manometer or continuously by the transducer method on 304D Siemens Sirecust monitor. Mid-axillary line was used as a reference point and 3—8 cmH_2O was taken as normal C.V.P. and as adequate hydration. The patient's urinary bladder was catheterized and urine output was monitored every 15 min. Chest X-rays were done in the post cooling period. Those patients in whom the central venous catheters were misplaced were dropped from this study. In our study, three patients had misplaced central venous catheters — all introduced from medial cubital veins (drum cartridge — Abbott).

RESULTS

Table I summarizes the data of all 34 patients at arrival and discharge from the cooling unit.

Twenty-two patients had C.V.P. of +3 cmH_2O or above (64.7%) and 12 patients had C.V.P. of zero or below zero cmH_2O (35.3%). Out of 22 patients with + C.V.P., 6 patients had C.V.P. of + 10 cmH_2O or more at the start of i.v. therapy (range 10—26 cmH_2O), Table II.

The mean volume of crystalloids infused was 1060 ml (ranges between 500 and 2500 ml) in an average cooling time of 1 h (range 20—96 min) (Table III). Only three patients needed more than 2 litres of fluid during cooling time.

The average time taken to normalize C.V.P in 12 low C.V.P. patients was 27 min (range being 5—50 min) (Table IV). Once the C.V.P. reached the normal level, rate of i.v. infusion was reduced to just keep the veins open. One patient (No. 15, Table I) normalized his C.V.P. within 5 min of cooling, another (No. 32, Table I) in 10 min after cooling in the post cooling unit. None of the patients developed overload problems such as CHF or pulmonary edema. All patients survived and left the hospital after 2—3 days.

DISCUSSION

During pilgrimage, most of the pilgrims coming from outside Saudi Arabia are not acclimatized to such hot, humid weather and the physical strain of the pilgrimage rituals consistently ranging for 5—7 days, may lead to dehydration, salt and water deficiency and any heat illness from prickly heat to heat syncope, heat exhaustion and sudden heat stroke. Muscular activity and rise in ambient temperature increase the metabolism and body temperature diverting the blood to exercising muscles and

Table I. The core temperature, blood pressure and C.V.P. at the time of arrival in heat stroke centre and duration of cooling, volume of i.v. infusion, volume of urine excreted and time to normalize C.V.P. during cooling period and C.V.P. on leaving the cooling unit is shown.

No.	Core temperature on arrival (°C)	B.P. on arrival (mmHg)	Initial C.V.P. (cmH_2O)	Cooling time (min)	Volume i.v. infusion (ml)	Time taken C.V.P. to normalize (min)	B.P. on leaving the unit (mmHg)	C.V.P. on leaving cooling unit (cmH_2O)	Urine output during cooling (ml)
1.	43.6	100/80	−2	96	2500	50	110/80	+6	650
2.	42.0	120/78	±3	82	1350	*	140/90	+5	220
3.	40.7	90/60	0	45	1000	20	110/70	+6	080
4.	42.3	160/100	+10	55	1080	*	155/110	+11	150
5.	40.9	140/90	+5	35	1000	*	155/100	+5	400
6.	42.8	145/90	+5	40	750	*	150/105	+7	110
7.	41.9	80/50	−4	65	2250	40	125/80	+6	280
8.	41.3	120/75	+7	50	1000	*	130/80	+9	160
9.	42.0	80/0	0	30	1000	30	120/100	+9	80
10.	42.8	130/80	+7	65	1750	*	150/90	+10	300
11.	43.0	90/40	0	75	2500	15	130/100	+8	440
12.	41.6	90/50	−1	40	1500	40	120/70	+4	180
13.	43.7	180/100	+10	100	1500	*	160/90	+9	670
14.	40.9	110/80	+6.5	25	500	*	175/100	+11	130
15.	41.5	130/70	+2	25	500	5	140/90	+12	70
16.	44.0	100/60	0	70	1200	20	115/80	+5	230
17.	43.6	90/50	−2	48	750	15	120/80	+3	50
18.	42.0	100/85	+16	55	600	*	110/70	+8	105
19.	42.6	110/90	+11	50	800	*	130/90	+10	190
20.	42.0	160/90	+3	38	1000	*	140/80	+6	95
21.	42.6	150/95	+8.5	40	750	*	155/100	+6	85
22.	42.0	120/70	+6	42	1100	*	135/80	+6	220
23.	41.8	106/80	+7	35	1000	*	125/90	+6	190
24.	40.2	180/110	+10	20	700	*	160/100	+10	110
25.	40.5	130/90	+4	70	900	*	140/85	+4	50
26.	42.5	125/70	+8	55	500	*	130/80	+6	80
27.	41.1	130/60	+6	35	750	*	140/80	+6	110
28.	43.0	80/0	0	70	1200	23	100/60	+5	100
29.	42.7	160/95	+12	50	900	*	150/90	+6	90
30.	41.6	100/60	−2	40	950	29	110/80	+6	85
31.	40.7	80/60	0	20	900	30	100/70	**	65
32.	42.5	115/80	+3	35	900	*	125/85	+5	240
33.	43.3	180/110	+12	43	500	*	185/100	+13	130
34.	42.7	140/75	+5	53	650	*	130/90	+7	75

*Had already normal C.V.P. and they were only monitored for any acute rise of C.V.P.
**C.V.P. normalized 10 min after transfer to post cooling unit.

Table II. Central venous pressure (cmH_2O) in 34 heat stroke patients on arrival at heat stroke centre.

C.V.P. (cmH_2O) ≤ 3	12	35.3%
C.V.P. (cmH_2O) ≥ 3—8	22	64.7%
C.V.P. (cmH_2O) ≥ 10	6	17.6%

Table III. Volume of I.V. fluid.

Mean volume of i.v. infusion	= 1060 ml
Maximum volume infused	= 2500 ml
Minimum volume infused	= 500 ml
Only in 3 patients volume infused exceeded	= 2000 ml

Table IV.

Mean time taken for C.V.P. to reach normal level (3—8 cmH_2O mid axillary lines) 12 patients	= 27.0 min
Range of time	= 5—50 min
S.D.	= 11.4855

the skin. This may lead to fall in cardiac output and hypotension may ensue and the victims of heat stroke may be deemed as fluid depleted and have been thus transfused briskly with a large volume of fluids.

The history and data of heat stroke victims collected by our group has shown that heat stroke prevails suddenly, sometimes without prodermal symptoms, and therefore these subjects should not always be considered as fluid depleted. In a majority of patients normal B.P., blood volume and cardiac output is found and some may even present with hyperkinetic circulation [3,8]. This is very well reflected by the volume of urine excretion during the cooling period when the patients were infused according to the C.V.P. (Table I). It is therefore perfectly plausible to predict that such patients if challenged with an unmonitored large volume of i.v. fluids, may develop acute cardiac failure and pulmonary edema, when they may not be fluid depleted. Even in the case of normo-volumic hypotension due to vasodilatation, during the process of cooling which may increase output of sympathomimetic amines and induce vasoconstriction, i.v. therapy with cold crystalloid solution room temperature (20—25°C) may normalize BP or even produce overload problems on the stressed C.V.S [8]. However, if hypotension persists and C.V.P. is high during the cooling process then thermic myocardial damage per se might have been the sequela to heat stroke. Cardiogenic shock due to myocardial infarction by no means is rare in heat stroke victims thus warranting again for careful fluid therapy.

The King Saud University heat stroke research team, after monitoring the haemodynamics of circulation with swan-ganz catheter in a 13-patient study, have similar results to our's [3]. On two occasions authors have used nitroprusside to dilate

the arteriolar bed to reduce the after load from the stressed heart due to very high pulmonary capillary wedge pressure. Incipient left ventricular failure could otherwise have easily produced catastrophic acute L.V.F. and pulmonary edema and its subsequent sequalae.

Surface cooling, induced cutaneous vaso constriction, and secretion of adrenaline and noradrenaline may increase the preload problem for the stressed C.V.S. Hypoxemia and hypercarbia may also attribute to rise in circulatory catecholamines [11—17]. The response to these sympathemimetic amines may differ from organ to organ as per temperature rise or fall during the cooling process and sympathetic discharge may have produced renal and splanchinic vasoconstriction leading to redistribution of the blood flow to vital autoregulatory organs like the brain. This stress on pulmonary capillary bed may also affect the right ventricle and may be the cause of acute rise in C.V.P. as may be seen in six patients. The sympathomimetic responses also increase afterload problems and thus pump failure may occur which may again lead to L.V.F. and pulmonary edema.

The insult of aspiration of the gastric contents in some patients whose consciousness is impaired might produce hypoxic pulmonary vasoconstriction [18]. All these factors may thus induce high pulmonary capillary wedge pressure which may be reflected on the left ventricle and central venous pressure.

We witnessed C.V.P. of more than 10 cmH_2O in six of our patients and the rest of the 22 patients who had normal range of C.V.P. could have reached very high C.V.P. if they had been challenged with a large volume of i.v. infusion at brisk rates. Thus there may always be a danger in developing overload problems in stressed C.V.S. and C.V.P. can be of great help in identifying the patients who may develop sudden acute ventricular failure and/or pulmonary edema if there should be a need for infusing large volumes of cold i.v. fluids.

In conclusion therefore, heat stroke victims should not be deemed as fluid depleted and should cautiously be challenged with i.v. fluids of more than a litre during the cooling period. Circulatory problems of heat stroke and pathophysiology of sympathetic discharge from the cooling process, hypoxemia, hypercarbia or inhalation injury and hypoxic pulmonary vasoconstriction leading to acute rise in C.V.P. has been discussed. Importance of establishing the C.V.P. line has been stressed to identify the patients who may not bear the insult of uncontrolled cold i.v. fluids injudiciously infused in a large quantity at a faster rate during the cooling time.

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Ambient Temperature and Mortality From Unintentional Cocaine Overdose

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Context.—Hot weather taxes cardiovascular function and is associated with increased deaths from heart disease. Cocaine can cause hypertension, tachycardia, coronary vasospasm, arrhythmias, and increased core temperature.

Objective.—To determine the association between mortality from cocaine overdose and hot weather.

Setting.—New York, NY.

Design.—Retrospective review of medical examiner cases from 1990 through 1995.

Subjects.—All fatal unintentional cocaine overdoses from 1990 through 1992 (n = 1382) and all hyperthermia deaths of cocaine users (n = 10) were used to identify a maximum daily temperature threshold above which mortality from cocaine intoxication increased. The study population consisted of all fatal unintentional cocaine overdoses from 1993 through 1995 (n = 2008) and 4 contemporaneous comparison groups that included fatal unintentional opiate overdoses (n = 793), all other fatal unintentional overdoses (n = 85), and a subset of homicides (n = 4638) and fatalities from motor vehicle crashes (n = 815).

Main Outcome Measures.—The number of overdose deaths and the proportion of homicides and traffic fatalities with a positive cocaine toxicology test result on days with a maximum temperature above or below the temperature threshold.

Results.—A threshold temperature of 31.1°C (88°F) was identified, above which the mean daily number of fatal cocaine overdoses increased steadily. On days with a maximum daily temperature of 31.1°C (88°F) or higher ("hot days"), the mean daily number of cocaine overdose deaths was 2.34 (SD = 1.68), which was 33% higher than the mean on days with a maximum temperature of less than 31.1°C (88°F) (mean = 1.76 [SD=1.37] (P<.001). In contrast, the mean number of opiate overdose deaths per day was 0.81 (SD = 0.94) on hot days and 0.71 (SD = 0.86) on other days (P = .28). For other drug overdose deaths, the mean number of deaths per day was 0.08 (SD = 0.28) on hot days and 0.08 (SD = 0.28) on other days (P = .69). Among homicides, the proportion with a positive cocaine toxicology test result was 18.9% on hot days and 19.5% on other days (P = .69), and among traffic fatalities, the proportions with positive cocaine toxicology test results were 9.5% on hot days and 10.3% on other days (P = .91).

Conclusions.—High ambient temperature is associated with a significant increase in mortality from cocaine overdose. Based on our comparison groups, the increase is not explained by changes in cocaine use among the general population. Although cocaine use is dangerous on all days, it appears to be even more dangerous on hot days.

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COCAINE-RELATED morbidity and mortality constitute a significant public health problem.[1] Approximately 8000 drug abuse deaths occur in the United States each year, about half of which involve cocaine use.[2] In addition, there are more than 400 000 emergency department visits for drug abuse, many of which involve cocaine.[3]

Cocaine use can result in serious toxic effects and death, in part because of hyperthermia.[4,5-7] However, even relatively low doses of cocaine can result in elevated core temperatures that are below those seen in extreme hyperthermia, but nonetheless tax cardiac reserve.[5,8] In addition, as a sympathomimetic agent, cocaine can produce tachycardia, hypertension, coronary vasospasm, and arrhythmias.[7-9] The association between cardiovascular deaths and hot weather is well documented.[10-18] Thus, the number of deaths from cocaine intoxication might be greater on days with high ambient temperature because of the effects of cocaine on cardiovascular function or its thermogenic properties.

For editorial comment see p 1828.

To investigate this hypothesis, we conducted a medical examiner surveillance study in New York City to assess the association of hot weather with mortality from unintentional cocaine overdoses. To rule out the possibility that a generalized increase of overdose deaths occurred on hot days, we also assessed the association of ambient temperature with fatal overdoses due to drugs other than cocaine, such as opiates, that do not have sympathomimetic or thermogenic properties.

The exact number of individuals who used cocaine on specific dates is unknown. Excess mortality from cocaine overdoses may occur on hot days, not because of the cardiovascular or thermogenic effects of the drug, but because more cocaine users may take the drug on such days. To examine this possibility, we also assessed the relationship between temperature and the presence of cocaine in 2 comparison groups, homicides and traffic fatalities.

METHODS

Fatal Unintentional Drug Overdoses

All cases of fatal unintentional drug overdoses in New York City from 1990 through 1995 were identified through manual review of all medical files at the Office of Chief Medical Examiner of New

York. This office has the responsibility for assessing all cases of persons believed to have died in an unnatural manner, including drug overdoses. To be classified as a subject, a person had to be certified as having died of an unintentional overdose caused by 1 or more drugs. Cases certified as suicidal or homicidal overdoses were excluded.

In the attribution of the cause and manner of death, the medical examiner uses the decedent's medical history, the circumstances and environment of the fatality, autopsy findings, and supporting laboratory data. A diagnosis of death caused by intoxication by 1 or more drugs requires that the toxicological data be within the range customarily encountered in such fatalities, that the history and circumstances be consistent with a fatal intoxication, and that the autopsy fail to disclose a disease or physical injury that has an extent or severity inconsistent with continued life. For instance, in the findings of a disease inconsistent with life, a concurrent intoxication would be regarded as incidental and unrelated to the cause of death.

In deaths determined to be caused by drug intoxication, when toxicology results reveal the presence of more than 1 drug in concentrations greater than trace amounts, it is customary to include all of the identified drugs in the cause of death. An exception occurs when 1 drug is present in extremely high concentrations and the others are present in concentrations commonly encountered in persons who die from other unequivocal causes, such as firearm injuries.

For purposes of data analysis, all fatal unintentional overdoses were divided into the following 3 hierarchical, mutually exclusive groups: (1) those in whom cocaine was, on the basis of history, circumstances, autopsy, and toxicological testing, causative of death; (2) those in whom opiates were causative of death, but in whom cocaine or its metabolite either were not detected or were detected in clinically insignificant amounts at toxicological testing; and (3) the remaining overdoses caused by substances other than cocaine or opiates.

For each case of fatal unintentional drug overdoses, demographic data, time, date, location of injury and death, cause and manner of death, and toxicology data were abstracted from the files. In a practical sense, the time of injury and of death differ by minutes to hours in almost all such cases.

Comparison Groups

We used 2 comparison groups of fatal injuries that occurred in the city from 1993 through 1995. Homicides were chosen as a comparison group because, on the whole, they have similar demographic characteristics to those dying of unintentional overdoses in New York City. Moreover, persons dying of homicide or unintentional drug overdoses generally reside in the same communities of the city that have high rates of drug use.[14] We also used a group composed of persons between the ages of 15 and 54 years who had died in a motor vehicle crash (as a driver, a passenger, or a pedestrian). This sample was restricted to this age group because 95% of cocaine overdoses occur among persons in this age range. In the same manner as in unintentional overdoses, for each homicide and traffic fatality, demographic data, time, date, location, cause and manner of death, and toxicology data were abstracted from the files.

Toxicologic Procedures

Urine and blood specimens collected at autopsy were stored at 4°C until they were assayed. A single toxicologic laboratory at the medical examiner's office performed all assays. Benzoylecgonine, the principal metabolite of cocaine, was initially screened for in urine by enzyme immunoassay. A specimen was considered positive if the concentration of benzoylecgonine was at least 0.3 mg/L. If urine was not readily available at autopsy, benzoylecgonine was screened for in blood by radioimmunoassay. A blood specimen was considered positive if the concentration of benzoylecgonine was at least 0.1 mg/L. Positive immunoassay findings were substantiated by radioimmunoassay in another tissue.

Blood was screened for cocaine by gas chromatography involving a nitrogen-phosphorus detector. A specimen was considered positive if the concentration of cocaine was equal to or exceeded 0.1 mg/L. All positive findings were confirmed by gas chromatography and mass spectrometry.

Opiates were screened for in urine by enzyme immunoassay or, if urine was not available, by radioimmunoassay in blood. Specimens that contained a concentration of at least 0.3 mg/L in urine or 0.1 mg in blood were considered positive, and were substantiated by radioimmunoassay in another tissue.

Head space chromatography was used to perform ethanol analysis. Other drugs were screened for and their presence confirmed by a variety of analytic methods, including enzyme immunoassay, thin-layer and high-performance liquid chromatography, and gas chromatography–mass spectrometry.

Temperature Data

We obtained hourly temperatures from the National Weather Service for Central Park Station in Manhattan, New York City, for each day from January 1, 1990, through December 31, 1995, and determined the daily maximum temperature in a 24-hour period. We hypothesized that the number of fatal overdoses involving cocaine would be higher on those days on which the ambient temperature exceeded an as yet undefined threshold temperature. Because we are unaware of any literature that has identified a temperature threshold for cocaine-related mortality, we sought to identify a threshold using our own data. As there is no single conventional method for determining such a threshold, we used the following strategy.

Sample of Hyperthermia Deaths That Involved Cocaine.—We identified all medical examiner cases from 1990 through 1995 that the medical examiner certified as due to hyperthermia (n = 48), but were not classified as unintentional drug overdoses. From this group, we identified all such cases in which benzoylecgonine was detected at autopsy (n = 10). The daily maximum temperatures for the 10 dates of death ranged from 17.2°C (63°F) to 39.4°C (103°F) (mean = 29.6°C [85.3°F], SD = 6.1°C [10.9°F], median = 30.9°C [87.5°F]).

Index Sample of Fatal Cocaine Overdoses.—Analyses of heat-related deaths often use a 1-day lag phase, which involves the comparison of mortality on days that exceed the threshold and the *next* day, with mortality on all other days.[15] For instance, a person who had died of an overdose may not have been found dead until the next day, or the date of pronounced death might have been recorded as the next day, especially if the death occurred just before midnight. We then examined the bivariate plot of the maximum daily temperature (x-axis) by the mean number of cocaine overdose deaths (y-axis) that occurred on days when the temperature was greater than or equal to a given temperature (Figure 1). For each temperature value, we determined the mean number of cocaine deaths occurring on days that had a maximum temperature that was greater than or equal to this specified temperature value. We plotted these data for the 1096 days from January 1, 1990, through December 31, 1992, which we refer to as the "index sample" and included all 1382 cases of fatal cocaine overdoses during this period (n = 1382). The plot shows a relatively flat relationship until 27.8°C (82°F), then a gradual increase, which accelerates more markedly at 31.1°C (88°F) (Figure 1).

Establishment of the Threshold Temperature.—Based on the median of the maximum temperature for the dates of deaths from the sample of hyperther-

mia cases involving cocaine and the plot of the index sample of fatal cocaine overdoses, we chose 31.1°C (88°F) as the threshold temperature. For purposes of analysis, we considered a day "hot" if the maximum hourly temperature was greater than or equal to 31.1°C (88°F), or if the maximum hourly temperature of the preceding day was greater than or equal to 31.1°C (88°F).

We considered reporting the results of analyses based on the heat index, which is a function of the temperature in degrees Fahrenheit and the relative humidity. However, for most persons the temperature is a more readily interpretable variable. Because temperature is a fundamental component of the heat index, the Pearson correlation between the daily maximum heat index and the daily maximum temperature in New York City during the study period was 0.986 ($P<.001$).

Statistical Analysis

All subsequent statistical analyses were performed using fatal unintentional overdoses that occurred during the next 3-year period, January 1, 1993, through December 31, 1995 (ie, the "cross-validation sample"). This split-sample approach to data analysis was used so that our hypothesis could be tested on a sample that was distinct from the index sample (1990-1992), which had been used to establish the threshold temperature.

Separate analyses were conducted for the 3 types of overdoses described earlier, deaths caused by cocaine; deaths caused by opiates, but not cocaine; and deaths caused by other drugs. The Mann-Whitney test was used to compare "hot" and "other" days with regard to the number of deaths from a particular type of overdose. Day was the unit of analysis. We also assessed the Pearson correlation coefficient for autocorrelation, which incorporated our 1-day lag phase.

We further examined whether cocaine use was increased on hot days among other causes of death during the same period, 1993 through 1995. For that reason, we compared the proportion of homicides and traffic-related fatalities with a positive cocaine toxicology on "hot" days with the respective proportions with a positive cocaine toxicology test result on "other" days. Using χ^2 tests, person was the unit of analysis.

For all analyses, 2-tailed α levels of .05 were considered statistically significant.

RESULTS

Deaths Due to Hyperthermia

During the 6-year period there were 48 deaths in New York City attributed to hyperthermia, including 34 males (70.8%) and 14 females (29.2%). Three (6.3%) of these deaths were among infants younger than 1 year, 29 (60.4%) were among those aged 15 through 54 years, and 16 (33.3%) were among those older than 54 years.

Among the 48 deaths, 40 (83.3%) were screened for benzoylecgonine and cocaine. Among these 40, benzoylecgonine was detected in 10 (25% of those screened, 21% of the total sample) and cocaine was detected in 7 (17.5% of those screened, 14.6% of the sample). In contrast, opiates were detected in only 1 case (2.5%) of the 40 screened. Ethanol was detected in only 5 (12.2%) of 41 cases screened.

When the sample of hyperthermia deaths was restricted to the age group that is most likely to have used cocaine (ie, those aged 15-54 years, n = 27), the percentages that were positive for benzoylecgonine and cocaine were 37.0% and 25.9%, respectively.

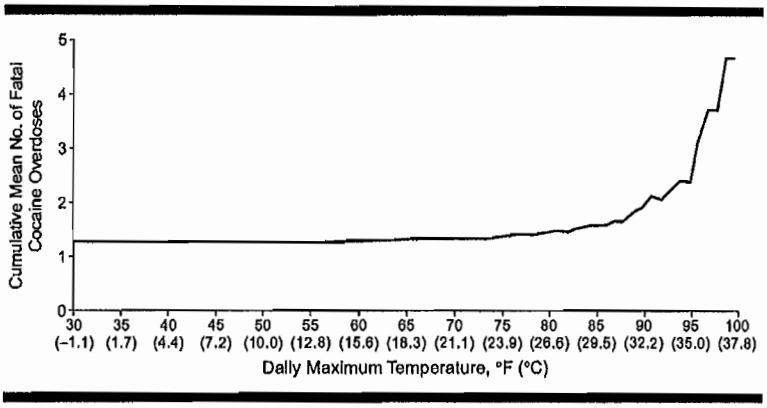

Figure 1.—Index sample. Mean number of fatal unintentional cocaine overdoses in New York City that occurred from 1990 through 1992 on days during which the maximum temperature was greater than or equal to each given temperature.

Table 1.—Number of Unintentional Deaths Due to Drug Overdoses, by Type of Day, Based on the Maximum Daily Temperature in New York City, January 1, 1993, Through December 31, 1995

Cause of Death	Type of Day*	No. of Deaths	Mean Daily No. of Deaths	SD	z†	P
Cocaine overdose (n = 2008)	Hot	341	2.34	1.68	3.98	<.001
	Other	1667	1.76	1.37		
Opiate overdose (n = 793)	Hot	118	0.81	0.94	1.08	.28
	Other	675	0.71	0.86		
Overdoses caused by drugs other than cocaine or opiates (n = 85)	Hot	12	0.08	0.28	0.39	.69
	Other	73	0.08	0.28		

*A day was considered "hot" if the maximum hourly temperature for that day or the preceding day was greater than or equal to 31.1°C (88°F). In the 3-year period, there were 1095 days, of which 146 days were considered hot and 949 days were below the temperature threshold of 31.1°C (88°F) (other).
†The z score from the normal approximation to the Mann-Whitney test.

Fatal Unintentional Cocaine Overdoses

There were 1095 days during the cross-validation period (1993-1995). Of these, 146 days (13.3%) were considered hot and 30 days were considered extremely hot, with maximum temperatures reaching 35°C (95°F) or higher. The hottest days of the period were July 15 and 16, 1995, when the maximum temperature was 39.4°C (103°F).

During this period, there were 2008 fatal unintentional cocaine overdoses. Complete autopsies were performed on 93% of these cases. The mean age of this group was 39.0 years (SD = 8.8 years), and 94.9% were aged 15 to 54 years. There were 1569 men (78.1%) and 439 women (21.9%), and 505 non-Hispanic whites (25.2%), 884 non-Hispanic blacks (44.0%), 605 Hispanics (30.1%), and 14 Asian or others (0.6%).

Significantly more deaths were due to cocaine overdoses on hot days (n = 146 hot days, mean = 2.34, SD = 1.68), than other days (n = 949 other days, mean = 1.76, SD = 1.37) (by Mann-Whitney, $z = 3.98$, $P<.001$) (Table 1). This difference represents a 33% increase in mean mortality on hot days. Mean daily mortality began to increase when the maximum temperature equalled or exceeded 31.1°C (88°F) (Figure 2). The 2 days with the highest number of cocaine overdose deaths (n = 9) were July 10 and 13, 1993, when the maximum temperatures were 37.2°C (99°F) and 33.3°C (92°F), respectively. There were no significant differences by age, race, gender, or location of death between subjects who had died on a hot day and those who had died on another day. The autocorrelation coefficient for cocaine overdose deaths was 0.12

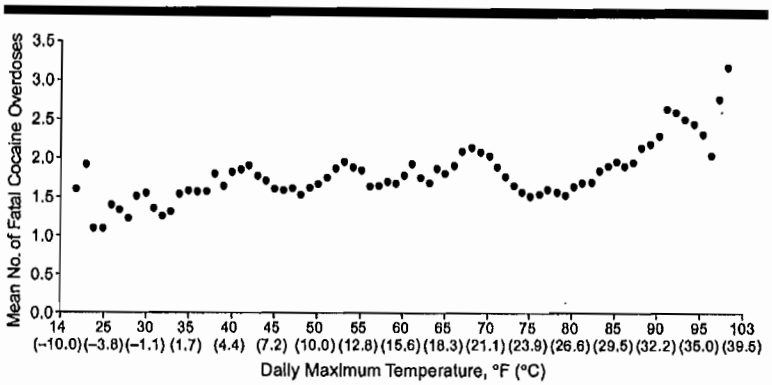

Figure 2.—Cross-validation sample. Mean number of fatal unintentional cocaine overdoses in New York City from 1993 through 1995 by the daily maximum temperature. The mean number of cocaine overdose deaths for a given temperature is plotted using centered moving averages spanning 5 degrees. (See the "Methods" section for more details about the use of a 1-day lag phase.)

Table 2.—Proportion of Fatal Cocaine Overdoses With a Positive Toxicology Result for Drugs Commonly Associated With a Risk of Hyperthermia*

	Cocaine Overdoses by Type of Day, % Positive	
Drug Type†	Hot (n = 341)	Other (n = 1667)
Antipsychotic medications‡	2.1	2.1
Antidepressant medications§	12.1	10.4
Antiparkinsonian and antihistaminic medications‖	5.0	9.7
Psychostimulants and sympathomimetic drugs¶	0.0	0.1
Phencyclidine	1.8	1.4
Any drug from above types	20.3	22.0

*In determining the percentage, an individual who had more than 1 drug of a given type detected (eg, an individual who had taken several different antidepressant medications) was counted only once for that drug type.
†Naming of the drug types reflects only the most common clinical indications for these classes of drugs.
‡Includes phenothiazines, thioxanthenes, dibenzodiazepines, butyrophenones, indolones, and diphenylbutylpiperidines.
§Includes tricyclic antidepressants and monoamine oxidase inhibitors.
‖Includes benztropine mesylate, biperiden, trihexyphenidyl hydrochloride, diphenhydramine hydrochloride, and chlorpheniramine maleate.
¶Includes amphetamines, methylphenidate hydrochloride, ephedrine, and phenylpropanolamine.

($P<.001$), which indicates that the number of cocaine overdoses on a prior day accounted for only 1.4% of the variance in cocaine overdose deaths.

Of the 341 subjects who died of cocaine overdoses on hot days, the proportion that had positive toxicology test results for ethanol was 42.9%, compared with 41.4% for the 1667 subjects who died on other days. Likewise, the proportions with positive toxicology test results for benzodiazepines, barbiturates, or salicylates on hot days were 10.3%, 1.2%, and 0.9%, respectively, compared with similar proportions as those who died on other days, which were 11.5% (benzodiazepines), 1.4% (barbiturates), and 1.3% (salicylates). Opiates were detected in 57.1% of cocaine overdose cases on hot days compared with 61.7% of such cases on other days.

Some drugs, including tricyclic antidepressants, antipsychotic medications, or psychostimulants, can affect heat regulation by central mechanisms, by increasing heat production through muscular activity, or by impairing heat dissipation. Many of these drugs have anticholinergic or sympathomimetic effects. Overall, only 20% of the individuals who died of cocaine overdoses had a positive toxicology test result for at least 1 of these drugs (Table 2), and the proportion of cocaine overdose deaths that had a positive toxicology test result for these agents on hot days was nearly identical to the proportion with a positive toxicology test result of those who died on other days (Table 2).

Fatal Unintentional Overdoses of Opiates and Other Drugs

During the cross-validation period, there were 793 fatal unintentional overdoses of opiates and 85 fatal overdoses that tested positive for neither cocaine nor opiates. There were no significant differences in the number of deaths on hot days and the number on other days for either of these drug-death groups (Table 1). Figure 3 and Figure 4 show no discernible change in the mean number of such deaths at any temperature.

Comparison Groups

During the cross-validation period, there were 4793 homicides in New York City, of which 4638 (96.8%) were assessed for the presence of cocaine in blood. Among these 4638 homicides, 902 homicides had a positive toxicology test result for cocaine. The demographic characteristics of the homicides that were positive for cocaine were similar to the cocaine overdose group (mean [SD] age = 32.9 [9.0] years; 97.5% between the ages of 15 and 54 years; 83.6% men; and 8.0% non-Hispanic white, 53.4% non-Hispanic black, 37.9% Hispanic, and 0.6% Asian or others). Of the 719 homicides that occurred on "hot" days, 136 (18.9%) had a positive toxicology test result for cocaine. Of the 3919 homicides that occurred on "other" days, 766 (19.5%) had a positive toxicology test result for cocaine ($\chi^2 = 0.11$; $df = 1$, $P = .69$).

During the cross-validation period, there were 921 traffic-related fatalities among persons between the ages of 15 and 54 years in New York City, of which 815 (88.5%) were assessed for cocaine in blood. Among these 815 fatalities, 83 had a positive toxicology test result for cocaine. The demographic characteristics of these 83 cases were similar to the cocaine overdose group (mean [SD] age = 34.4 [8.5] years; 79.5% men; and 14.5% non-Hispanic white, 44.5% non-Hispanic black, 39.8% Hispanic, and 1.2% Asian or others). Of the 126 traffic fatalities that occurred on "hot" days, 12 (9.5%) had a positive toxicology test result for cocaine. Of the 689 traffic fatalities that occurred on "other" days, 71 (10.3%) had a positive toxicology test result for cocaine ($\chi^2 = 0.01$, $df = 1$, $P = .91$).

COMMENT

This study demonstrates several main findings. First, a quarter of all individuals younger than 55 years who died from hyperthermia in New York City had used cocaine immediately prior to their deaths. Second, the mean number of deaths from cocaine overdoses on days in which the temperature was equal to or greater than 31.1°C (88°F) was 33% higher than the mean on days with lower maximum temperatures. On days with temperatures higher than 31.1°C (88°F), daily mortality from cocaine overdoses increased steadily. Third, the association of ambient temperature with drug overdose mortality appears specific to cocaine, and not to other major drugs of abuse, such as opiates. Although the number of deaths from cocaine overdose was increased on hot days, only 10% of days in a year on average reach a temperature of 31.1°C (88°F) in New York City. Thus, the temperature effect we

found does not have a major influence on the annual mortality from cocaine overdoses.

The exact reason for an association between high ambient temperature with mortality from cocaine overdose is not known. Increased temperature places considerable demands on the cardiovascular system to increase cardiac output and to decrease systemic vascular resistance.[16] The immediate effects of cocaine use include increases in mean arterial pressure, heart rate, and cardiac output.[17] Moreover, cocaine use has been associated with cardiac abnormalities including coronary artery occlusion, malignant arrhythmias, and myocarditis.[9] Thus, cocaine use during hot weather may further tax cardiovascular capacity and increase the risk of mortality.

Another explanation is that the excess of such deaths on hot days may, in some cases, be due to the thermogenic effects of cocaine. These effects have been postulated to result from the propensity of cocaine to cause increased muscular activity and peripheral vasoconstriction, or its direct effect on dopamine-modulated, heat-regulatory centers in the hypothalamus.[18-20] For instance, several case reports have documented fatal hyperthermia with and without rhabdomyolosis in cocaine users.[21-23] Dogs administered intravenous cocaine experienced a significant increase in core temperatures, which proved fatal.[24,25] Moreover, above an ambient temperature of 11.5°C (52.7°F) the increase in core body temperature in canines was positively correlated with the ambient temperature at which the cocaine had been administered.[25]

Hyperthermia is a clinical diagnosis, rather than a diagnosis established by pathologic findings. Therefore, the number of deaths classified with this cause is quite limited. Persons who overdose on cocaine may not be discovered for hours after death, long after it would have been possible to determine if a clinical hyperthermia syndrome had preceded their deaths. Although we cannot determine from our database whether many of the fatal cocaine overdoses on hot days were attributable to hyperthermia, at least 1 in 4 deaths in New York known to have resulted from hyperthermia had a positive cocaine toxicology test result. This prevalence is considerably greater than the 1.3% prevalence of recent cocaine use (in the past 30 days) reported in general population household surveys of drug use in New York.[26] Moreover, most cocaine-related hyperthermia deaths occurred on warm or hot days. It is possible that some of the deaths from acute cocaine intoxication may have been cases of undetected hyperthermia.

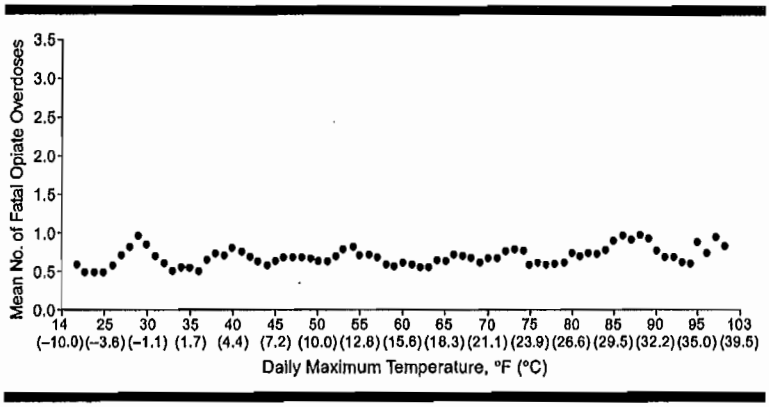

Figure 3.—Mean number of fatal unintentional opiate overdoses (not caused by cocaine) by the maximum daily temperature for the cross-validation period, 1993 through 1995, in New York City. The mean number of opiate overdose deaths for a given temperature is plotted using centered moving averages spanning 5 degrees. (See the "Methods" section for more details about the use of a 1-day lag phase.)

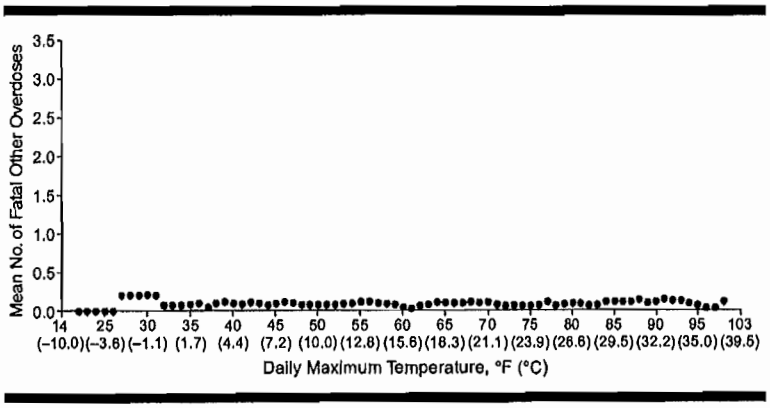

Figure 4.—Mean number of fatal unintentional overdoses caused by drugs other than cocaine or opiates by the maximum daily temperature for the cross-validation period, 1992 through 1995, in New York City. The mean number of overdose deaths caused by drugs other than cocaine or opiates for a given temperature is plotted using centered moving averages spanning 5 degrees. (See the "Methods" section for more details about the use of a 1-day lag phase.)

Drug overdose deaths that involve a single agent are uncommon. Many cocaine users concurrently administer opiates, ethanol, and other sedative drugs. However, only about 20% of cocaine overdose cases had a positive toxicology test result for other drugs that are commonly associated with a risk of hyperthermia. Moreover, the proportions of cocaine overdose deaths with a toxicology test result positive for at least 1 of these drugs was virtually the same for those who died on hot days vs other days. Thus, our findings cannot be attributed exclusively to the presence of other drugs that impair heat regulation. It is probable that individuals who had administered cocaine along with anticholinergic or sympathomimetic agents may have enhanced their risk of heat-related mortality.

Our findings cannot be explained by a generalized increase in the number of overdose deaths on hot days as we observed no association of ambient temperature with overdoses caused by opiates or by other drugs. It is also unlikely that our findings can be explained by an increase in cocaine use in the New York City population on hot days. Among homicides, which have similar demographic characteristics and reside in similar neighborhoods as cocaine overdose cases, and among persons involved in motor vehicle crashes, we found that cocaine was detected in virtually identical proportions among those dying on hot days or on other days. Rates of screening for drugs also are not influenced by hot weather in New York City.

It is likely that our findings from New York would apply to other cities in the United States. Ambient temperatures in cities are often higher than other regions because of increased heat production from crowds, cars, and factories; re-

tention of heat by buildings and pavements; and diminished heat loss because of low wind speed.[27,28] Socioeconomic factors also may contribute to heat-related mortality, as city residents are likely to be older and poorer, and more likely to live in substandard housing without air conditioning, showers, or baths, than suburban or rural residents.[29] In an earlier study, we found that many drug overdose deaths occur in poor neighborhoods,[14] which are likely to include few homes with air conditioners.

Cities in the Northeast and on the Pacific Coast have the lowest threshold temperatures above which general mortality increases.[30] People who reside in higher latitudes or near oceans experience fewer hot days, but have a harder time adjusting to sudden changes in temperature, particularly in early summer.[10,27] Thus, the threshold of 31.1°C (88°F) that we found in New York may vary somewhat in other locations.

Typically, periods of high humidity and warm nighttime temperatures, which reflect persistent, high heat exposure and diminished cooling from evaporative loss, have been associated with increased general mortality, rather than transient daytime peaks in temperature.[31] However, the models that best account for variability in general mortality in New York City involve dry, hot conditions (ie, a threshold of 33.3°C [92°F], a low dew point, and a 1-day lag phase in mortality).[30] Our analyses using the heat index yielded similar results, which is not surprising given that during the study period the maximum daily heat index was so highly correlated with the maximum daily temperature.

Several methodologic issues in our study warrant comment. First, it is not possible to know the dose of cocaine that an individual had used prior to death. Thus, the relationship between dose of cocaine and ambient temperature is unknown. Second, we do not know the exact ambient temperature to which the subjects were exposed when they used cocaine. However, even nighttime low temperatures on very hot days in New York remain in the 70s. Third, the threshold temperature of 31.1°C (88°F) was established for a group and not for individuals. Some individuals may have higher or lower thresholds at which their mortality risk from cocaine use changes. Fourth, we do not know whether the individuals who died were occasional cocaine users or long-term, heavy users.

Although it is not possible to prove that high ambient temperature results in a direct, increased risk of death from cocaine use, our data suggest that the increased mortality from cocaine overdoses on hot days is not explained by changes in cocaine use in the general population on such days. Cocaine use is dangerous on all days. Our findings suggest that the risk of death associated with cocaine increases further during hot weather.

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criteria for a recommended standard . . . .

OCCUPATIONAL EXPOSURE TO HOT ENVIRONMENTS

Revised Criteria 1986

NIOSH

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

Criteria for a Recommended Standard....

Occupational Exposure to Hot Environments

Revised Criteria 1986

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health
Division of Standards Development and Technology Transfer

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FOREWORD

The Occupational Safety and Health Act of 1970 (Public Law 91-596) states that the purpose of Congress expressed in the Act is "to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources...by," among other things, "providing medical criteria which will assure insofar as practicable that no worker will suffer diminished health, functional capacity, or life expectancy as a result of his work experience." In the Act, the National Institute for Occupational Safety and Health (NIOSH) is authorized to "develop and establish recommended occupational safety and health standards..." and to "conduct such research and experimental programs as...are necessary for the development of criteria for new and improved occupational safety and health standards..."

The Institute responds to these mandates by means of the criteria document. The essential and distinguishing feature of a criteria document is that it recommends a standard for promulgation by an appropriate regulatory body, usually the Occupational Safety and Health Administration (OSHA) or the Mine Safety and Health Administration (MSHA) of the U.S. Department of Labor. NIOSH is also responsible for reviewing existing OSHA and MSHA standards and previous recommendations by NIOSH, to ensure that they are adequate to protect workers in view of the current state of knowledge. Updating criteria documents, when necessary, is an essential element of that process.

A criteria document, Criteria for a Recommended Standard....Occupational Exposure to Hot Environments, was prepared in 1972. The current revision presented here takes into account the vast amount of new scientific information on working in hot environments which is pertinent to safety and health. Included are ways of predicting the health risks, procedures for control of heat stress, and techniques for prevention and treatment of heat-related illnesses.

External review consultants drawn from academia, business associations, labor organizations, private consultants, and representatives of other governmental agencies, contributed greatly to the form and content of this revised document. However, responsibility for the conclusions reached and the recommendations made, belongs solely to the Institute. All comments by reviewers, whether or not incorporated into the document are being sent with it to the Occupational Safety and Health Administration (OSHA) for consideration in standard setting.

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I. RECOMMENDATIONS FOR AN OCCUPATIONAL STANDARD FOR WORKERS EXPOSED TO HOT ENVIRONMENTS

The National Institute for Occupational Safety and Health (NIOSH) recommends that worker exposure to heat stress in the workplace be controlled by complying with all sections of the recommended standard found in this document. This recommended standard should prevent or greatly reduce the risk of adverse health effects to exposed workers and will be subject to review and revision as necessary.

Heat-induced occupational illnesses, injuries, and reduced productivity occur in situations in which the total heat load (environmental plus metabolic) exceeds the capacities of the body to maintain normal body functions without excessive strain. The reduction of adverse health effects can be accomplished by the proper application of engineering and work practice controls, worker training and acclimatization, measurements and assessment of heat stress, medical supervision, and proper use of heat-protective clothing and equipment.

In this criteria document, total heat stress is considered to be the sum of heat generated in the body (metabolic heat) plus the heat gained from the environment (environmental heat) minus the heat lost from the body to the environment. The bodily response to total heat stress is called the heat strain. Many of the bodily responses to heat exposure are desirable and beneficial. However, at some level of heat stress, the worker's compensatory mechanisms will no longer be capable of maintaining body temperature at the level required for normal body functions. As a result, the risk of heat-induced illnesses, disorders, and accidents substantially increases. The level of heat stress at which excessive heat strain will result depends on the heat-tolerance capabilities of the worker. However, even though there is a wide range of heat tolerance between workers, each worker has an upper limit for heat stress beyond which the resulting heat strain can cause the worker to become a heat casualty. In most workers, appropriate repeated exposure to elevated heat stress causes a series of physiologic adaptations called acclimatization, whereby the body becomes more efficient in coping with the heat stress. Such an acclimatized worker can tolerate a greater heat stress before a harmful level of heat strain occurs.

The occurrence of heat-induced illnesses and unsafe acts among a group of workers in a hot environment, or the recurrence of such problems in, individual workers, represents "sentinel health events" (SHE's) which indicate that heat control measures, medical screening, or environmental monitoring measures may not be adequate [1]. One or more occurrences of heat-induced illness in a particular worker indicates the need for medical inquiry about the possibility of temporary or permanent loss of the worker's ability to tolerate heat stress. The recommended requirements in the following sections are intended to establish the permissible limits of total heat stress so that the risk of incurring heat-induced illnesses and disorders in workers is reduced.

Almost all healthy workers, who are not acclimatized to working in hot environments and who are exposed to combinations of environmental and metabolic heat less than the appropriate NIOSH Recommended Alert Limits (RAL's) given in Figure 1, should be able to tolerate total heat without substantially increasing their risk of incurring acute adverse health effects. Almost all healthy workers, who are heat-acclimatized to working in hot environments and who are exposed to combinations of environmental and metabolic heat less than the appropriate NIOSH Recommended Exposure Limits (REL's) given in Figure 2, should be capable of tolerating the total heat without incurring adverse effects. The estimates of both environmental and metabolic heat are expressed as 1-hour time-weighted averages (TWAs) as described in reference [2].

At combinations of environmental and metabolic heat exceeding the Ceiling Limits (C) in Figures 1 and 2, no worker shall be exposed without adequate heat-protective clothing and equipment. To determine total heat loads where a worker could not achieve thermal balance, but might sustain up to a 1 degree Celsius (1°C) rise in body temperature in less than 15 minutes, the Ceiling Limits were calculated using the heat balance equation given in Chapter III, Section A.

In this criteria document, healthy workers are defined as those who are not excluded from placement in hot environment jobs by the explicit criteria given in Chapters I, IV, VI, and VII. These exclusionary criteria are qualitative in that the epidemiologic parameters of sensitivity, specificity, and predictive power of the evaluation methods are not fully documented. However, the recommended exclusionary criteria represent the best judgment of NIOSH based on the best available data and comments of peer reviewers. This may include both absolute and relative exclusionary indicators related to age, stature, gender, percent body fat, medical and occupational history, specific chronic diseases or therapeutic regimens, and the results of such tests as the maximum aerobic capacity ($\dot{V}O_2max$), electrocardiogram (EKG), pulmonary function tests (PFTs), and chest x rays (CXRs).

The medical surveillance program shall be designed and implemented in such a way as to minimize the risk of the workers' health and safety being jeopardized by any heat hazards that may be present in the workplace (see Chapters IV, VI, and VII). The medical program shall provide for both preplacement medical examinations for those persons who are candidates for a hot job and periodic medical examinations for those workers who are currently working in hot jobs.

Section 1 - Workplace Limits and Surveillance

(a) Recommended Limits

(1) Unacclimatized workers: Total heat exposure to workers shall be controlled so that unprotected healthy workers who are not acclimatized to working in hot environments are not exposed to combinations of metabolic and environmental heat greater than the applicable RAL's given in Figure 1.

(2) Acclimatized workers: Total heat exposure to workers shall be controlled so that unprotected healthy workers who are acclimatized to working in hot environments are not exposed to combinations of metabolic and environmental heat greater than the applicable REL's given in Figure 2.

(3) Effect of Clothing: The recommended limits given in Figures 1 and 2 are for healthy workers who are physically and medically fit for the level of activity required by their job and who are wearing the customary one layer work clothing ensemble consisting of not more than long-sleeved work shirts and trousers (or equivalent). The REL and RAL values given in Figures 1 and 2 may not provide adequate protection if workers wear clothing with lower air and vapor permeability or insulation values greater than those for the customary one layer work clothing ensemble discussed above. A discussion of these modifications to the REL and RAL is given in Chapter III, Section C.

(4) Ceiling Limits: No worker shall be exposed to combinations of metabolic and environmental heat exceeding the applicable Ceiling Limits (C) of Figures 1 or 2 without being provided with and properly using appropriate and adequate heat-protective clothing and equipment.

(b) Determination of Environmental Heat

(1) Measurement methods: Environmental heat exposures shall be assessed by the Wet Bulb Globe Thermometer (WBGT) method or equivalent techniques, such as Effective Temperature (ET), Corrected Effective Temperature (CET), or Wet Globe Temperature (WGT), that can be converted to WBGT values (as described in Chapters V and IX). The WBGT shall be accepted as the standard method and its readings the standard against which all others are compared. When air- and vapor-impermeable protective clothing is worn, the dry bulb temperature (t_a) or the adjusted dry bulb temperature (t_{adb}) is a more appropriate measurement.

(2) Measurement requirements: Environmental heat measurements shall be made at or as close as feasible to the work area where the worker is exposed. When a worker is not continuously exposed in a single hot area, but moves between two or more areas with differing levels of environmental heat or when the environmental heat substantially varies at the single hot area, the environmental heat exposures shall be measured at each area and during each period of constant heat levels where employees are exposed. Hourly TWA WBGTs shall be calculated for the combination of jobs (tasks), including all scheduled and unscheduled rest periods.

(3) Modifications of work conditions: Environmental heat measurements shall be made at least hourly during the hottest portion of each workshift, during the hottest months of the year, and when a heat wave occurs or is predicted. If two such sequential measurements exceed the applicable RAL or REL, then work

conditions shall be modified by use of appropriate engineering controls, work practices, or other measures until two sequential measures are in compliance with the exposure limits of this recommended standard.

(4) Initiation of measurements: A WBGT or an individual environmental factors profile shall be established for each hot work area for both winter and summer seasons as a guide for determining when engineering controls and/or work practices or other control methods shall be instituted. After the environmental profiles have been established, measurements shall be made as described in (b)(1), (2), and (3) of this section during the time of year and days when the profile indicates that total heat exposures above the applicable RAL's or REL's may be reasonably anticipated or when a heat wave has been forecast by the nearest National Weather Service station or other competent weather forecasting service.

(c) Determination of Metabolic Heat

(1) Metabolic heat screening estimates: For initial screening purposes, metabolic heat rates for each worker shall either be measured as required in (c)(2) of this section or shall be estimated from Table V-3 to determine whether the total heat exposure exceeds the applicable RAL or REL. For determination of metabolic heat, Table V-3 shall be used only for screening purposes unless other reliable and valid baseline data have been developed and confirmed by the indirect open-circuit method specified in (c)(2) of this Section. When computing metabolic heat estimates using Table V-3 for screening purposes, the metabolic heat production in kilocalories per minute shall be calculated using the upper value of the range given in Table V-3 for each body position and type of work for each specific task(s) of each worker's job.

EXAMPLE:

As shown in Table V-3 (D, Sample calculation), for a task that requires the worker to stand and use both arms, the values to be added would be 0.6 kilocalories per minute (kcal/min) for standing, 3.5 kcal/min for working with both arms, and 1.0 kcal/min for basal metabolism, for a total metabolic heat of 5.1 kcal/min for a worker who weighs 70 kilograms (kg)(154 lb). For a worker that has other than a 70-kg weight, the metabolic heat shall be corrected by the factor (actual worker weight in kg/70 kg). Thus for an 85-kg worker the factor would be (85/70) = 1.21 and the appropriate estimate for metabolic heat would be (1.21)(5.1) = 6.2 kcal/min for the duration of the task.

(2) Metabolic heat measurements - Whenever the combination of measured environmental heat (WBGT) and screening estimate of metabolic heat exceeds the applicable RAL or REL (Figures 1 and 2), the metabolic heat production shall be measured using the indirect open-circuit procedure (see Chapter V) or an equivalent method.

Metabolic heat rates shall be expressed as kilocalories per hour (kcal/h), British thermal units (Btu) per hour, or watts (W) for a 1-hour TWA task basis that includes all activities engaged in during each period of analysis and all scheduled and nonscheduled rest periods (1 kcal/h = 3.97 Btu/h = 1.16 W).

EXAMPLE:

For the example in (c)(1), if the task was performed by an acclimatized 70-kg worker for the entire 60 minutes of each hour, the screening estimate for the 1-hour TWA metabolic heat would be (5.1 kcal/min)(60 min) = about 300 kcal/h. Using the applicable Figure 2, a vertical line at 300 kcal/h would intersect the 60 min/h REL curve at a WBGT of 27.8°C (82°F). Then, if the measured WBGT exceeds 27.8°C, proceed to measure the worker's metabolic heat with the indirect open-circuit method or equivalent procedure.

If the 70-kg worker was unacclimatized, use of Figure 1 indicates that metabolic heat measurement of the worker would be required above a WBGT of 25°C (77°F).

(d) Physiologic Monitoring

Physiologic monitoring may be used as an adjunct monitoring procedure to those estimates and measurements required in the preceding Parts (a), (b), and (c) of this section. The total heat stress shall be considered to exceed the applicable RAL or REL when the physiologic functions (e.g., core or oral body temperature, work and recovery pulse rate) exceed the values given in Chapter IX, Section D.

Section 2 - Medical Surveillance

(a) General

(1) The employer shall institute a medical surveillance program for all workers who are or may be exposed to heat stress above the RAL, whether they are acclimatized or not.

(2) The employer shall assure that all medical examinations and procedures are performed by or under the direction of a licensed physician.

(3) The employer shall provide the required medical surveillance without cost to the workers, without loss of pay, and at a reasonable time and place.

(b) Preplacement Medical Examinations

For the purposes of the preplacement medical examination, all workers shall be considered to be unacclimatized to hot environments. At a minimum, the preplacement medical examination of each prospective worker for a hot job shall include:

(1) A comprehensive work and medical history, with special emphasis on any medical records or information concerning any known or suspected previous heat illnesses or heat intolerance. The medical history shall contain relevant information on the cardiovascular system, skin, liver, kidney, and the nervous and respiratory systems;

(2) A comprehensive physical examination that gives special attention to the cardiovascular system, skin, liver, kidney, and the nervous and respiratory systems;

(3) An assessment of the use of therapeutic drugs, over-the-counter medications, or social drugs (including alcohol), that may increase the risk of heat injury or illness (see Chapter VII);

(4) An assessment of obesity (body fatness), that is defined as exceeding 25% of normal weight for males and exceeding 30% of normal weight for females, as based on age and body build;

(5) An assessment of the worker's ability to wear and use any protective clothing and equipment, especially respirators, that is or may be required to be worn or used; and

(6) Other factors and examination details included in Chapter VII, Section B-1.

(c) Periodic Medical Examinations

Periodic medical examinations shall be made available at least annually to all workers who may be exposed at the worksite to heat stress exceeding the RAL. The employer shall provide the examinations specified in Part (b) above including any other items the examining physician considers relevant. If circumstances warrant (e.g., increase in job-related heat stress, changes in health status), the medical examination shall be offered at shorter intervals at the discretion of the responsible physician.

(d) Emergency Medical Care

If the worker for any reason develops signs or symptoms of heat illness, the employer shall provide appropriate emergency medical treatment.

(e) Information to be Provided to the Physician

The employer shall provide the following information to the examining physician performing or responsible for the medical surveillance program:

(1) A copy of this recommended standard;

(2) A description of the affected worker's duties and activities as they relate to the worker's environmental and metabolic heat exposure;

(3) An estimate of the worker's potential exposure to workplace heat (both environmental and metabolic), including any available workplace measurements or estimates;

(4) A description of any protective equipment or clothing the worker uses or may be required to use; and

(5) Relevant information from previous medical examinations of the affected worker which is not readily available to the examining physician.

(f) Physician's Written Opinion

The employer shall obtain a written opinion from the responsible physician which shall include:

(1) The results of the medical examination and the tests performed;

(2) The physician's opinion as to whether the worker has any detected medical conditions which would increase the risk of material impairment of health from exposure to anticipated heat stress in the work environment;

(3) An estimate of the individual's tolerance to withstand hot working conditions;

(4) An opinion as to whether the worker can perform the work required by the job (i.e., physical fitness for the job);

(5) Any recommended limitations upon the worker's exposure to heat stress or upon the use of protective clothing or equipment; and

(6) A statement that the worker has been informed by the physician of the results of the medical examination and any medical conditions which require further explanation or treatment.

The employer shall provide a copy of the physician's written opinion to the affected worker.

Section 3 - Surveillance of Heat-Induced Sentinel Health Events

(a) Definition

Surveillance of heat-induced Sentinel Health Events (SHE's) is defined as the systematic collection and analysis of data concerning the occurrence and distribution of adverse health effects in defined populations at risk to heat injury or illness.

(b) Requirements

In order to evaluate and improve prevention and control measures for heat-induced effects, which includes the identification of highly susceptible workers, data on the occurrence or recurrence in the same

worker, and distribution in time, place, and person of heat-induced adverse effects shall be obtained and analyzed for each workplace.

Section 4 - Posting of Hazardous Areas

(a) Dangerous Heat-Stress Areas

In work areas and at entrances to work areas or building enclosures where there is a reasonable likelihood of the combination(s) of environmental and metabolic heat exceeding the Ceiling Limit, there shall be posted readily visible warning signs containing information on the required protective clothing or equipment, hazardous effects of heat stress on human health, and information on emergency measures for heat injury or illness. This information shall be arranged as follows:

DANGEROUS HEAT-STRESS AREA
HEAT-STRESS PROTECTIVE CLOTHING OR EQUIPMENT REQUIRED
HARMFUL IF EXCESSIVE HEAT EXPOSURE OR WORK LOAD OCCUR
HEAT-INDUCED FAINTING, HEAT EXHAUSTION, HEAT CRAMP,
HEAT RASH OR HEAT STROKE MAY OCCUR

(b) Emergency Situations

In any area where there is a likelihood of heat stress emergency situations occurring, the warning signs required in (a) of this section shall be supplemented with signs giving emergency and first aid instructions.

(c) Additional Requirements for Warning Signs

All hazard warning signs shall be printed in English and where appropriate in the predominant language of workers unable to read English. Workers unable to read the signs shall be informed of the warning printed on the signs and the extent of the hazardous area(s). All warning signs shall be kept clean and legible at all times.

Section 5 - Protective Clothing and Equipment

Engineering controls and safe work practices shall be used to maintain worker exposure to heat stress at or below the applicable RAL or REL specified in Section 1. In addition, protective clothing and equipment (e.g., water-cooled garments, air-cooled garments, ice-packet vests, wetted-overgarments, heat-reflective aprons or suits) shall be provided by the employer to the workers when the total heat stress exceeds the Ceiling Limit.

Section 6 - Worker Information and Training

(a) Information Requirements

All new and current workers, who are unacclimatized to heat and work in areas where there is reasonable likelihood of heat injury or illness, shall be kept informed, through continuing education programs, of:

(1) Heat stress hazards,

(2) Predisposing factors and relevant signs and symptoms of heat injury and illness,

(3) Potential health effects of excessive heat stress and first aid procedures,

(4) Proper precautions for work in heat stress areas,

(5) Worker responsibilities for following proper work practices and control procedures to help protect the health and provide for the safety of themselves and their fellow workers, including instructions to immediately report to the employer the development of signs or symptoms of heat stress overexposure,

(6) The effects of therapeutic drugs, over-the-counter medications, or social drugs (including alcohol), that may increase the risk of heat injury or illness by reducing heat tolerance (see Chapter VII),

(7) The purposes for and descriptions of the environmental and medical surveillance programs and of the advantages to the worker of participating in these surveillance programs, and

(8) If necessary, proper use of protective clothing and equipment.

(b) Continuing Education Programs

(1) The employer shall institute a continuing education program, conducted by persons qualified by experience or training in occupational safety and health, to ensure that all workers potentially exposed to heat stress have current knowledge of at least the information specified in (a) of this section. For each affected worker, the instructional program shall include adequate verbal and/or written communication of the specified information. The employer shall develop a written plan of the training program that includes a record of all instructional materials.

(2) The employer shall inform all affected workers of the location of written training materials and shall make these materials readily available, without cost to the affected workers.

(c) Heat-Stress Safety Data Sheet

(1) The information specified in (a) of this section shall be recorded on a heat-stress safety data sheet or on a form specified by the Occupational Safety and Health Administration (OSHA).

(2) In addition, the safety data sheet shall contain:

(i) Emergency and first aid procedures, and

(ii) Notes to physician regarding classification, medical aspects, and prevention of heat injury and illness. These notes shall include information on the category and clinical features of each injury and illness, predisposing factors, underlying physiologic disturbance, treatment, and prevention procedures (see Table IV-1).

Section 7 - Control of Heat Stress

(a) General Requirements

(1) Where engineering and work practice controls are not sufficient to reduce exposures to or below the applicable RAL or REL, they shall, nonetheless, be used to reduce exposures to the lowest level achievable by these controls and shall be supplemented by the use of heat-protective clothing or equipment, and a heat-alert program shall be implemented as specified in (d) of this section.

(2) The employer shall establish and implement a written program to reduce exposures to or below the applicable RAL or REL by means of engineering and work practice controls.

(b) Engineering Controls

(1) The type and extent of engineering controls required to bring the environmental heat below the applicable RAL or REL can be calculated using the basic heat exchange formulae (e.g., Chapters III and VI). When the environmental heat exceeds the applicable RAL or REL, the following control requirements shall be used.

(a) When the air temperature exceeds the skin temperature, convective heat gain shall be reduced by decreasing air temperature and/or decreasing the air velocity if it exceeds 1.5 meters per second (m/sec) (300 ft/min). When air temperature is lower than skin temperature, convective heat loss shall be increased by increasing air velocity. The type, amount, and characteristics of clothing will influence heat exchange between the body and the environment.

(b) When the temperature of the surrounding solid objects exceeds skin temperature, radiative heat gain shall be reduced by: placing shielding or barriers, that are radiant-reflecting or heat-absorbing, between the heat source and the worker; by isolating the source of radiant heat; or by modifying the hot process or operation.

(c) When necessary, evaporative heat loss shall be increased by increasing air movement over the worker, by reducing the influx of moisture from steam leaks or from water on the workplace floors, or by reducing the water vapor content (humidity) of the air. The air and water vapor permeability of the clothing worn by the worker will influence the rate of heat exchange by evaporation.

(c) Work and Hygienic Practices

(1) Work modifications and hygienic practices shall be introduced to reduce both environmental and metabolic heat when engineering controls are not adequate or are not feasible. The most effective preventive work and hygienic practices for reducing heat stress include, but are not limited to the following parts of this section:

(a) Limiting the time the worker spends each day in the hot environment by decreasing exposure time in the hot environment and/or increasing recovery time spent in a cool environment;

(b) Reducing the metabolic demands of the job by such procedures as mechanization, use of special tools, or increase in the number of workers per task;

(c) Increasing heat tolerance by a heat acclimatization program and by increasing physical fitness;

(d) Training supervisors and workers to recognize early signs and symptoms of heat illnesses and to administer relevant first aid procedures;

(e) Implementing a buddy system in which workers are responsible for observing fellow workers for early signs and symptoms of heat intolerance such as weakness, unsteady gait, irritability, disorientation, changes in skin color, or general malaise; and

(f) Providing adequate amounts of cool, i.e., 10° to 15°C (50° to 59°F) potable water near the work area and encouraging all workers to drink a cup of water (about 150 to 200 mL (5 to 7 ounces) every 15 to 20 minutes. Individual, not communal, drinking cups shall be provided.

(d) Heat-Alert Program

A written Heat-Alert Program shall be developed and implemented whenever the National Weather Service or other competent weather forecast service forecasts that a heat wave is likely to occur the following day or days. A heat wave is indicated when daily maximum temperature exceeds 35°C (95°F) or when the daily maximum temperature exceeds 32°C (90°F) and is 5°C (9°F) or more above the maximum reached on the preceding days. The details for a Heat-Alert Program are described in Chapter VI, Section C.

Section 8 - Recordkeeping

(a) Environmental Surveillance

(1) The employer shall establish and maintain an accurate record of all measurements made to determine environmental and metabolic

heat exposures to workers as required in Section 1 of this recommended standard.

(2) Where the employer has determined that no metabolic heat measurements are required as specified in Section 1, Part (c)(2) of this recommended standard, the employer shall maintain a record of the screening estimates relied upon to reach the determination.

(b) Medical Surveillance

The employer shall establish and maintain an accurate record for each worker subject to medical surveillance as specified in Section 2 of this recommended standard.

(c) Surveillance of Heat-Induced Sentinel Health Events

The employer shall establish and maintain an accurate record of the data and analyses specified in Section 3 of this recommended standard.

(d) Heat-Induced Illness Surveillance

The employer shall establish and maintain an accurate record of any heat-induced illness or injury and the environmental and work conditions at the time of the illness or injury.

(e) Heat Stress Tolerance Augmentation

The employer shall establish and maintain an accurate record of all heat stress tolerance augmentation for workers by heat acclimatization procedures and/or physical fitness enhancement.

(f) Record Retention

In accordance with the requirements of 29 CFR 1910.20(d), the employer shall retain records described by this recommended standard for at least the following periods:

(1) Thirty years for environmental monitoring records,

(2) Duration of employment plus 30 years for medical surveillance records,

(3) Thirty years for surveillance records for heat-induced SHE's, and

(4) Thirty years for records of heat stress tolerance augmentation

(g) Availability of Records

(1) The employer shall make worker environmental surveillance records available upon request for examination and copying to the subject worker or former worker or to anyone having the specific

written consent of the subject worker or former worker in accordance with 29 CFR 1910.20.

(2) Any worker's medical surveillance records, surveillance records for heat-induced SHE's, or records of heat stress tolerance augmentation that are required by this recommended standard shall be provided upon request for examination and copying to the subject worker or former worker or to anyone having the specific written consent of the subject worker or former worker.

(h) Transfer of Records

(1) The employer shall comply with the requirements on the transfer of records set forth in the standard, Access to Medical Records, 29 CFR 1910.20(h).

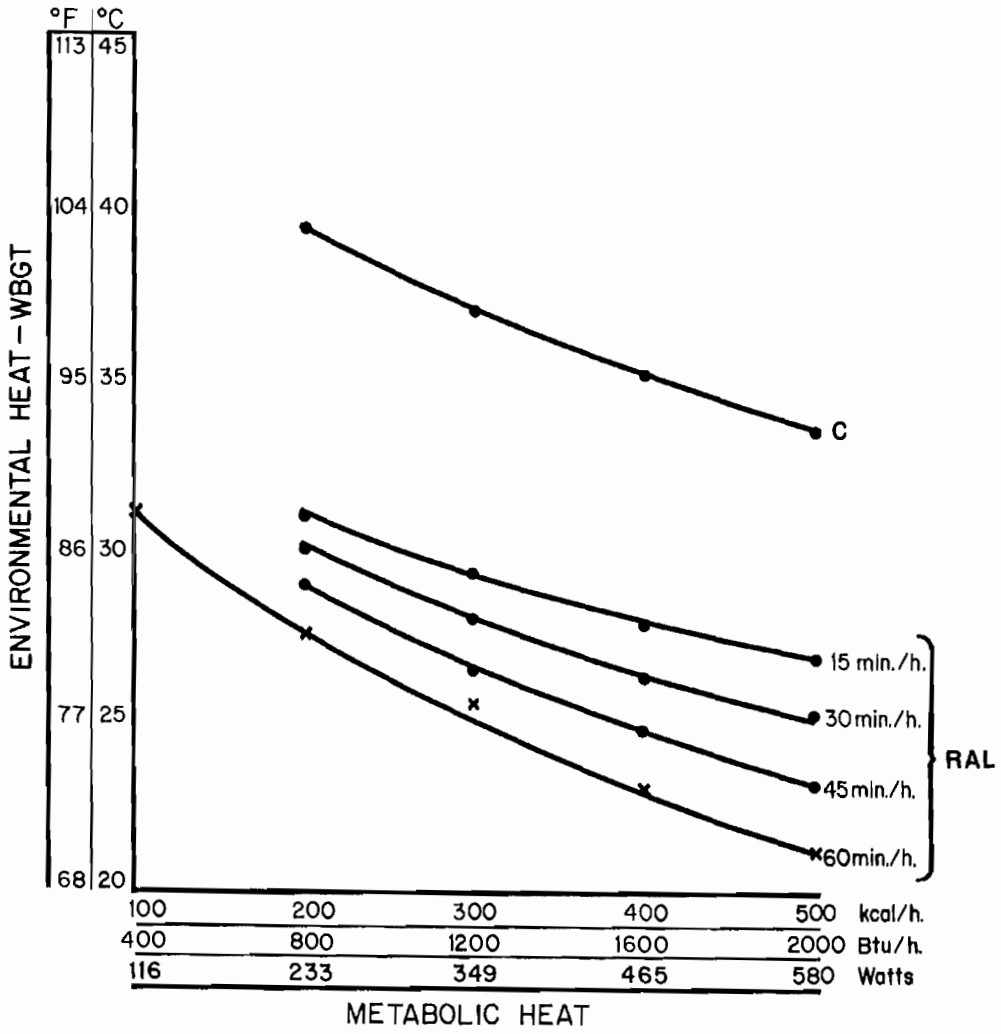

Figure 1. Recommended Heat-Stress Alert Limits
Heat-Unacclimatized Workers

C = Ceiling Limit
RAL = Recommended Alert Limit
*For "standard worker" of 70 kg (154 lbs) body weight and 1.8 m^2 (19.4 ft^2) body surface.
Based on References 2,3,4,5,6,7,8.

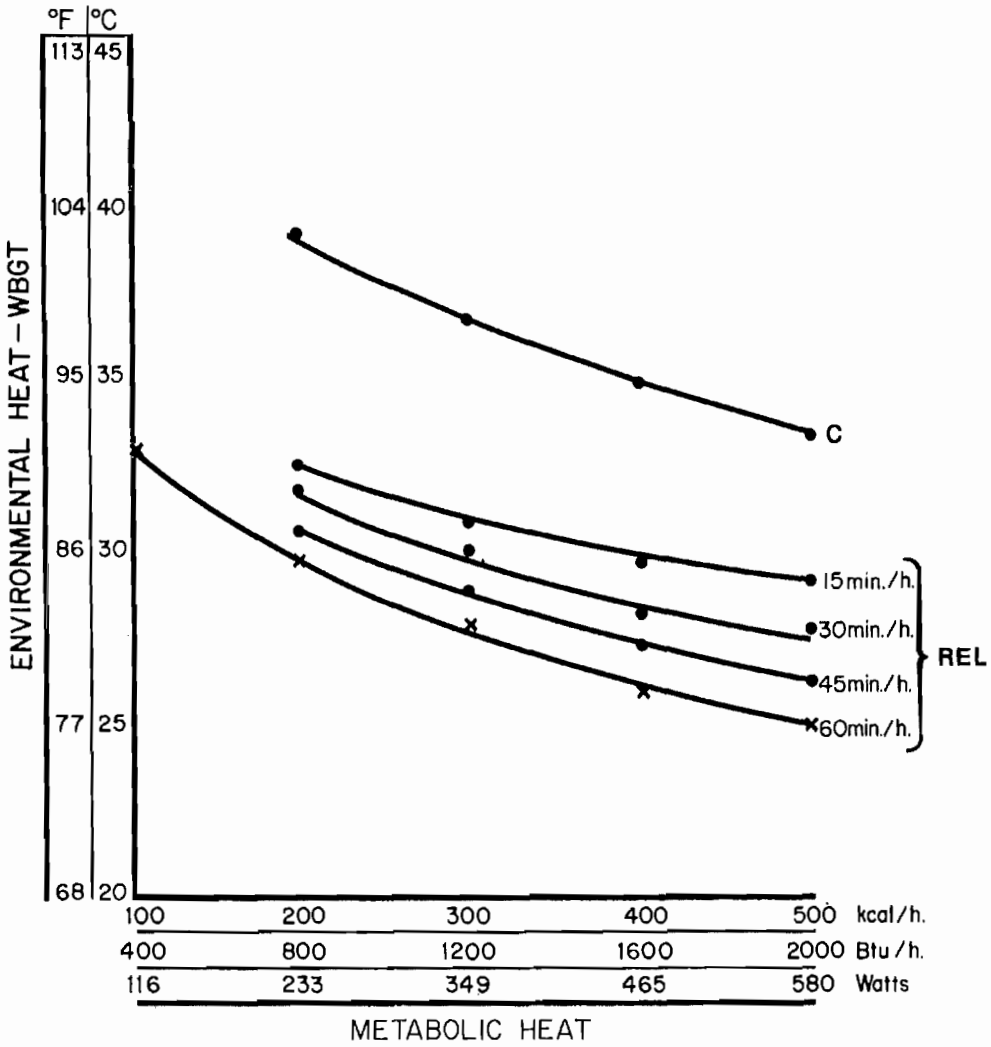

Figure 2. Recommended Heat-Stress Exposure Limits
Heat-Acclimatized Workers

C = Ceiling Limit
REL = Recommended Exposure Limit
*For "standard worker" of 70 kg (154 lbs) body weight and 1.8 m^2 (19.4 ft^2) body surface.
Based on References 2,3,4,5,6,7,8.

II. INTRODUCTION

Criteria documents are developed by the National Institute for Occupational Safety and Health (NIOSH), in response to section 20(a)(3) of the Occupational Safety and Health Act of 1970. In the Act, NIOSH is charged with developing criteria documents for toxic chemical substances and harmful physical agents which will describe exposure levels that are safe for various periods of employment including but not limited to the exposure levels at which no worker will suffer impaired health or functional capacities or diminished life expectancy as a result of any work experience. Environmental heat is a potentially harmful physical agent. This document presents the criteria and recommendations for a standard that were prepared to meet the need for preventing heat-induced health impairment resulting from exposure to occupational heat stress.

This document is an update of the Criteria for a Recommended Standard.... Occupational Exposure to Hot Environments (HSM-10269) published by NIOSH in January 1972 [9]. In June 1972, NIOSH sent the criteria document to the Occupational Safety and Health Administration (OSHA). In January 1973, the Assistant Secretary of Labor for Occupational Safety and Health appointed a 15 member Standards Advisory Committee on Heat Stress to review the NIOSH criteria document and develop a proposed standard. The committee submitted a proposed standard to the Assistant Secretary of Labor, OSHA, in January 1974 [7]. A standard on occupational exposure to hot environments was not promulgated. The updating of this document is based on the relevant scientific data and industry experience that have accrued since the original document was prepared. The document presents the criteria, techniques, and procedures for the assessment, evaluation, and control of occupational heat stress by engineering and preventive work practices and those for the recognition, treatment, and prevention of heat-induced illnesses and unsafe acts by medical supervision, hygienic practices, and training programs.

The recommended criteria were developed to ensure that adherence to them will (1) protect against the risk of heat-induced illnesses and unsafe acts, (2) be achievable by techniques that are valid, reproducible, and available, and (3) be attainable by existing techniques. This recommended standard is also designed to prevent possible harmful effects from interactions between heat and toxic chemical and physical agents. The recommended environmental limits for various intensities of physical work as indicated in Figures 1 and 2 are not upper tolerance limits for heat exposure for all workers but rather levels at which engineering controls, preventive work and hygienic practices, and administrative or other control procedures should be implemented in order to reduce the risk of heat illnesses even in the least heat-tolerant workers.

Estimates of the number of industrial workers who are exposed to heat stress on the job are at best rough guesses. A review of the Statistical Abstracts of the United States, 105th edition 1985, for the number of workers in industries where heat stress is a potential safety and health hazard indicates that a conservative estimate would be 5 to 10 million workers [10].

A glossary of terms, symbols, abbreviations, and units of measure used in this document is presented in XII-A.

criteria for a recommended standard . . . .

OCCUPATIONAL EXPOSURE TO HOT ENVIRONMENTS

Revised Criteria 1986

NIOSH

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

Criteria for a Recommended Standard....

Occupational Exposure to Hot Environments

Revised Criteria 1986

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health
Division of Standards Development and Technology Transfer

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DISCLAIMER

Mention of the name of any company or product does not constitute endorsement by the National Institute for Occupational Safety and Health.

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FOREWORD

The Occupational Safety and Health Act of 1970 (Public Law 91-596) states that the purpose of Congress expressed in the Act is "to assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources...by," among other things, "providing medical criteria which will assure insofar as practicable that no worker will suffer diminished health, functional capacity, or life expectancy as a result of his work experience." In the Act, the National Institute for Occupational Safety and Health (NIOSH) is authorized to "develop and establish recommended occupational safety and health standards..." and to "conduct such research and experimental programs as...are necessary for the development of criteria for new and improved occupational safety and health standards..."

The Institute responds to these mandates by means of the criteria document. The essential and distinguishing feature of a criteria document is that it recommends a standard for promulgation by an appropriate regulatory body, usually the Occupational Safety and Health Administration (OSHA) or the Mine Safety and Health Administration (MSHA) of the U.S. Department of Labor. NIOSH is also responsible for reviewing existing OSHA and MSHA standards and previous recommendations by NIOSH, to ensure that they are adequate to protect workers in view of the current state of knowledge. Updating criteria documents, when necessary, is an essential element of that process.

A criteria document, Criteria for a Recommended Standard....Occupational Exposure to Hot Environments, was prepared in 1972. The current revision presented here takes into account the vast amount of new scientific information on working in hot environments which is pertinent to safety and health. Included are ways of predicting the health risks, procedures for control of heat stress, and techniques for prevention and treatment of heat-related illnesses.

External review consultants drawn from academia, business associations, labor organizations, private consultants, and representatives of other governmental agencies, contributed greatly to the form and content of this revised document. However, responsibility for the conclusions reached and the recommendations made, belongs solely to the Institute. All comments by reviewers, whether or not incorporated into the document are being sent with it to the Occupational Safety and Health Administration (OSHA) for consideration in standard setting.

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The National Institute for Occupational Safety and Health (NIOSH) recommends that worker exposure to heat stress in the workplace be controlled by complying with all sections of the recommended standard found in this document. This recommended standard should prevent or greatly reduce the risk of adverse health effects to exposed workers and will be subject to review and revision as necessary.

Heat-induced occupational illnesses, injuries, and reduced productivity occur in situations in which the total heat load (environmental plus metabolic) exceeds the capacities of the body to maintain normal body functions without excessive strain. The reduction of adverse health effects can be accomplished by the proper application of engineering and work practice controls, worker training and acclimatization, measurements and assessment of heat stress, medical supervision, and proper use of heat-protective clothing and equipment.

In this criteria document, total heat stress is considered to be the sum of heat generated in the body (metabolic heat) plus the heat gained from the environment (environmental heat) minus the heat lost from the body to the environment. The bodily response to total heat stress is called the heat strain. Many of the bodily responses to heat exposure are desirable and beneficial. However, at some level of heat stress, the worker's compensatory mechanisms will no longer be capable of maintaining body temperature at the level required for normal body functions. As a result, the risk of heat-induced illnesses, disorders, and accidents substantially increases. The level of heat stress at which excessive heat strain will result depends on the heat-tolerance capabilities of the worker. However, even though there is a wide range of heat tolerance between workers, each worker has an upper limit for heat stress beyond which the resulting heat strain can cause the worker to become a heat casualty. In most workers, appropriate repeated exposure to elevated heat stress causes a series of physiologic adaptations called acclimatization, whereby the body becomes more efficient in coping with the heat stress. Such an acclimatized worker can tolerate a greater heat stress before a harmful level of heat strain occurs.

The occurrence of heat-induced illnesses and unsafe acts among a group of workers in a hot environment, or the recurrence of such problems in, individual workers, represents "sentinel health events" (SHE's) which indicate that heat control measures, medical screening, or environmental monitoring measures may not be adequate [1]. One or more occurrences of heat-induced illness in a particular worker indicates the need for medical inquiry about the possibility of temporary or permanent loss of the worker's ability to tolerate heat stress. The recommended requirements in the following sections are intended to establish the permissible limits of total heat stress so that the risk of incurring heat-induced illnesses and disorders in workers is reduced.

Almost all healthy workers, who are not acclimatized to working in hot environments and who are exposed to combinations of environmental and metabolic heat less than the appropriate NIOSH Recommended Alert Limits (RAL's) given in Figure 1, should be able to tolerate total heat without substantially increasing their risk of incurring acute adverse health effects. Almost all healthy workers, who are heat-acclimatized to working in hot environments and who are exposed to combinations of environmental and metabolic heat less than the appropriate NIOSH Recommended Exposure Limits (REL's) given in Figure 2, should be capable of tolerating the total heat without incurring adverse effects. The estimates of both environmental and metabolic heat are expressed as 1-hour time-weighted averages (TWAs) as described in reference [2].

At combinations of environmental and metabolic heat exceeding the Ceiling Limits (C) in Figures 1 and 2, no worker shall be exposed without adequate heat-protective clothing and equipment. To determine total heat loads where a worker could not achieve thermal balance, but might sustain up to a 1 degree Celsius (1°C) rise in body temperature in less than 15 minutes, the Ceiling Limits were calculated using the heat balance equation given in Chapter III, Section A.

In this criteria document, healthy workers are defined as those who are not excluded from placement in hot environment jobs by the explicit criteria given in Chapters I, IV, VI, and VII. These exclusionary criteria are qualitative in that the epidemiologic parameters of sensitivity, specificity, and predictive power of the evaluation methods are not fully documented. However, the recommended exclusionary criteria represent the best judgment of NIOSH based on the best available data and comments of peer reviewers. This may include both absolute and relative exclusionary indicators related to age, stature, gender, percent body fat, medical and occupational history, specific chronic diseases or therapeutic regimens, and the results of such tests as the maximum aerobic capacity ($\dot{V}O_2max$), electrocardiogram (EKG), pulmonary function tests (PFTs), and chest x rays (CXRs).

The medical surveillance program shall be designed and implemented in such a way as to minimize the risk of the workers' health and safety being jeopardized by any heat hazards that may be present in the workplace (see Chapters IV, VI, and VII). The medical program shall provide for both preplacement medical examinations for those persons who are candidates for a hot job and periodic medical examinations for those workers who are currently working in hot jobs.

Section 1 - Workplace Limits and Surveillance

(a) Recommended Limits

(1) Unacclimatized workers: Total heat exposure to workers shall be controlled so that unprotected healthy workers who are not acclimatized to working in hot environments are not exposed to combinations of metabolic and environmental heat greater than the applicable RAL's given in Figure 1.

(2) Acclimatized workers: Total heat exposure to workers shall be controlled so that unprotected healthy workers who are acclimatized to working in hot environments are not exposed to combinations of metabolic and environmental heat greater than the applicable REL's given in Figure 2.

(3) Effect of Clothing: The recommended limits given in Figures 1 and 2 are for healthy workers who are physically and medically fit for the level of activity required by their job and who are wearing the customary one layer work clothing ensemble consisting of not more than long-sleeved work shirts and trousers (or equivalent). The REL and RAL values given in Figures 1 and 2 may not provide adequate protection if workers wear clothing with lower air and vapor permeability or insulation values greater than those for the customary one layer work clothing ensemble discussed above. A discussion of these modifications to the REL and RAL is given in Chapter III, Section C.

(4) Ceiling Limits: No worker shall be exposed to combinations of metabolic and environmental heat exceeding the applicable Ceiling Limits (C) of Figures 1 or 2 without being provided with and properly using appropriate and adequate heat-protective clothing and equipment.

(b) Determination of Environmental Heat

(1) Measurement methods: Environmental heat exposures shall be assessed by the Wet Bulb Globe Thermometer (WBGT) method or equivalent techniques, such as Effective Temperature (ET), Corrected Effective Temperature (CET), or Wet Globe Temperature (WGT), that can be converted to WBGT values (as described in Chapters V and IX). The WBGT shall be accepted as the standard method and its readings the standard against which all others are compared. When air- and vapor-impermeable protective clothing is worn, the dry bulb temperature (t_a) or the adjusted dry bulb temperature (t_{adb}) is a more appropriate measurement.

(2) Measurement requirements: Environmental heat measurements shall be made at or as close as feasible to the work area where the worker is exposed. When a worker is not continuously exposed in a single hot area, but moves between two or more areas with differing levels of environmental heat or when the environmental heat substantially varies at the single hot area, the environmental heat exposures shall be measured at each area and during each period of constant heat levels where employees are exposed. Hourly TWA WBGTs shall be calculated for the combination of jobs (tasks), including all scheduled and unscheduled rest periods.

(3) Modifications of work conditions: Environmental heat measurements shall be made at least hourly during the hottest portion of each workshift, during the hottest months of the year, and when a heat wave occurs or is predicted. If two such sequential measurements exceed the applicable RAL or REL, then work

conditions shall be modified by use of appropriate engineering controls, work practices, or other measures until two sequential measures are in compliance with the exposure limits of this recommended standard.

(4) Initiation of measurements: A WBGT or an individual environmental factors profile shall be established for each hot work area for both winter and summer seasons as a guide for determining when engineering controls and/or work practices or other control methods shall be instituted. After the environmental profiles have been established, measurements shall be made as described in (b)(1), (2), and (3) of this section during the time of year and days when the profile indicates that total heat exposures above the applicable RAL's or REL's may be reasonably anticipated or when a heat wave has been forecast by the nearest National Weather Service station or other competent weather forecasting service.

(c) Determination of Metabolic Heat

(1) Metabolic heat screening estimates: For initial screening purposes, metabolic heat rates for each worker shall either be measured as required in (c)(2) of this section or shall be estimated from Table V-3 to determine whether the total heat exposure exceeds the applicable RAL or REL. For determination of metabolic heat, Table V-3 shall be used only for screening purposes unless other reliable and valid baseline data have been developed and confirmed by the indirect open-circuit method specified in (c)(2) of this Section. When computing metabolic heat estimates using Table V-3 for screening purposes, the metabolic heat production in kilocalories per minute shall be calculated using the upper value of the range given in Table V-3 for each body position and type of work for each specific task(s) of each worker's job.

EXAMPLE:

As shown in Table V-3 (D, Sample calculation), for a task that requires the worker to stand and use both arms, the values to be added would be 0.6 kilocalories per minute (kcal/min) for standing, 3.5 kcal/min for working with both arms, and 1.0 kcal/min for basal metabolism, for a total metabolic heat of 5.1 kcal/min for a worker who weighs 70 kilograms (kg)(154 lb). For a worker that has other than a 70-kg weight, the metabolic heat shall be corrected by the factor (actual worker weight in kg/70 kg). Thus for an 85-kg worker the factor would be (85/70) = 1.21 and the appropriate estimate for metabolic heat would be (1.21)(5.1) = 6.2 kcal/min for the duration of the task.

(2) Metabolic heat measurements - Whenever the combination of measured environmental heat (WBGT) and screening estimate of metabolic heat exceeds the applicable RAL or REL (Figures 1 and 2), the metabolic heat production shall be measured using the indirect open-circuit procedure (see Chapter V) or an equivalent method.

Metabolic heat rates shall be expressed as kilocalories per hour (kcal/h), British thermal units (Btu) per hour, or watts (W) for a 1-hour TWA task basis that includes all activities engaged in during each period of analysis and all scheduled and nonscheduled rest periods (1 kcal/h = 3.97 Btu/h = 1.16 W).

EXAMPLE:

For the example in (c)(1), if the task was performed by an acclimatized 70-kg worker for the entire 60 minutes of each hour, the screening estimate for the 1-hour TWA metabolic heat would be (5.1 kcal/min)(60 min) = about 300 kcal/h. Using the applicable Figure 2, a vertical line at 300 kcal/h would intersect the 60 min/h REL curve at a WBGT of 27.8°C (82°F). Then, if the measured WBGT exceeds 27.8°C, proceed to measure the worker's metabolic heat with the indirect open-circuit method or equivalent procedure.

If the 70-kg worker was unacclimatized, use of Figure 1 indicates that metabolic heat measurement of the worker would be required above a WBGT of 25°C (77°F).

(d) Physiologic Monitoring

Physiologic monitoring may be used as an adjunct monitoring procedure to those estimates and measurements required in the preceding Parts (a), (b), and (c) of this section. The total heat stress shall be considered to exceed the applicable RAL or REL when the physiologic functions (e.g., core or oral body temperature, work and recovery pulse rate) exceed the values given in Chapter IX, Section D.

Section 2 - Medical Surveillance

(a) General

(1) The employer shall institute a medical surveillance program for all workers who are or may be exposed to heat stress above the RAL, whether they are acclimatized or not.

(2) The employer shall assure that all medical examinations and procedures are performed by or under the direction of a licensed physician.

(3) The employer shall provide the required medical surveillance without cost to the workers, without loss of pay, and at a reasonable time and place.

(b) Preplacement Medical Examinations

For the purposes of the preplacement medical examination, all workers shall be considered to be unacclimatized to hot environments. At a minimum, the preplacement medical examination of each prospective worker for a hot job shall include:

(1) A comprehensive work and medical history, with special emphasis on any medical records or information concerning any known or suspected previous heat illnesses or heat intolerance. The medical history shall contain relevant information on the cardiovascular system, skin, liver, kidney, and the nervous and respiratory systems;

(2) A comprehensive physical examination that gives special attention to the cardiovascular system, skin, liver, kidney, and the nervous and respiratory systems;

(3) An assessment of the use of therapeutic drugs, over-the-counter medications, or social drugs (including alcohol), that may increase the risk of heat injury or illness (see Chapter VII);

(4) An assessment of obesity (body fatness), that is defined as exceeding 25% of normal weight for males and exceeding 30% of normal weight for females, as based on age and body build;

(5) An assessment of the worker's ability to wear and use any protective clothing and equipment, especially respirators, that is or may be required to be worn or used; and

(6) Other factors and examination details included in Chapter VII, Section B-1.

(c) Periodic Medical Examinations

Periodic medical examinations shall be made available at least annually to all workers who may be exposed at the worksite to heat stress exceeding the RAL. The employer shall provide the examinations specified in Part (b) above including any other items the examining physician considers relevant. If circumstances warrant (e.g., increase in job-related heat stress, changes in health status), the medical examination shall be offered at shorter intervals at the discretion of the responsible physician.

(d) Emergency Medical Care

If the worker for any reason develops signs or symptoms of heat illness, the employer shall provide appropriate emergency medical treatment.

(e) Information to be Provided to the Physician

The employer shall provide the following information to the examining physician performing or responsible for the medical surveillance program:

(1) A copy of this recommended standard;

(2) A description of the affected worker's duties and activities as they relate to the worker's environmental and metabolic heat exposure;

(3) An estimate of the worker's potential exposure to workplace heat (both environmental and metabolic), including any available workplace measurements or estimates;

(4) A description of any protective equipment or clothing the worker uses or may be required to use; and

(5) Relevant information from previous medical examinations of the affected worker which is not readily available to the examining physician.

(f) Physician's Written Opinion

The employer shall obtain a written opinion from the responsible physician which shall include:

(1) The results of the medical examination and the tests performed;

(2) The physician's opinion as to whether the worker has any detected medical conditions which would increase the risk of material impairment of health from exposure to anticipated heat stress in the work environment;

(3) An estimate of the individual's tolerance to withstand hot working conditions;

(4) An opinion as to whether the worker can perform the work required by the job (i.e., physical fitness for the job);

(5) Any recommended limitations upon the worker's exposure to heat stress or upon the use of protective clothing or equipment; and

(6) A statement that the worker has been informed by the physician of the results of the medical examination and any medical conditions which require further explanation or treatment.

The employer shall provide a copy of the physician's written opinion to the affected worker.

Section 3 - Surveillance of Heat-Induced Sentinel Health Events

(a) Definition

Surveillance of heat-induced Sentinel Health Events (SHE's) is defined as the systematic collection and analysis of data concerning the occurrence and distribution of adverse health effects in defined populations at risk to heat injury or illness.

(b) Requirements

In order to evaluate and improve prevention and control measures for heat-induced effects, which includes the identification of highly susceptible workers, data on the occurrence or recurrence in the same

worker, and distribution in time, place, and person of heat-induced adverse effects shall be obtained and analyzed for each workplace.

Section 4 - Posting of Hazardous Areas

(a) Dangerous Heat-Stress Areas

In work areas and at entrances to work areas or building enclosures where there is a reasonable likelihood of the combination(s) of environmental and metabolic heat exceeding the Ceiling Limit, there shall be posted readily visible warning signs containing information on the required protective clothing or equipment, hazardous effects of heat stress on human health, and information on emergency measures for heat injury or illness. This information shall be arranged as follows:

DANGEROUS HEAT-STRESS AREA
HEAT-STRESS PROTECTIVE CLOTHING OR EQUIPMENT REQUIRED
HARMFUL IF EXCESSIVE HEAT EXPOSURE OR WORK LOAD OCCUR
HEAT-INDUCED FAINTING, HEAT EXHAUSTION, HEAT CRAMP,
HEAT RASH OR HEAT STROKE MAY OCCUR

(b) Emergency Situations

In any area where there is a likelihood of heat stress emergency situations occurring, the warning signs required in (a) of this section shall be supplemented with signs giving emergency and first aid instructions.

(c) Additional Requirements for Warning Signs

All hazard warning signs shall be printed in English and where appropriate in the predominant language of workers unable to read English. Workers unable to read the signs shall be informed of the warning printed on the signs and the extent of the hazardous area(s). All warning signs shall be kept clean and legible at all times.

Section 5 - Protective Clothing and Equipment

Engineering controls and safe work practices shall be used to maintain worker exposure to heat stress at or below the applicable RAL or REL specified in Section 1. In addition, protective clothing and equipment (e.g., water-cooled garments, air-cooled garments, ice-packet vests, wetted-overgarments, heat-reflective aprons or suits) shall be provided by the employer to the workers when the total heat stress exceeds the Ceiling Limit.

Section 6 - Worker Information and Training

(a) Information Requirements

All new and current workers, who are unacclimatized to heat and work in areas where there is reasonable likelihood of heat injury or illness, shall be kept informed, through continuing education programs, of:

(1) Heat stress hazards,

(2) Predisposing factors and relevant signs and symptoms of heat injury and illness,

(3) Potential health effects of excessive heat stress and first aid procedures,

(4) Proper precautions for work in heat stress areas,

(5) Worker responsibilities for following proper work practices and control procedures to help protect the health and provide for the safety of themselves and their fellow workers, including instructions to immediately report to the employer the development of signs or symptoms of heat stress overexposure,

(6) The effects of therapeutic drugs, over-the-counter medications, or social drugs (including alcohol), that may increase the risk of heat injury or illness by reducing heat tolerance (see Chapter VII),

(7) The purposes for and descriptions of the environmental and medical surveillance programs and of the advantages to the worker of participating in these surveillance programs, and

(8) If necessary, proper use of protective clothing and equipment.

(b) Continuing Education Programs

(1) The employer shall institute a continuing education program, conducted by persons qualified by experience or training in occupational safety and health, to ensure that all workers potentially exposed to heat stress have current knowledge of at least the information specified in (a) of this section. For each affected worker, the instructional program shall include adequate verbal and/or written communication of the specified information. The employer shall develop a written plan of the training program that includes a record of all instructional materials.

(2) The employer shall inform all affected workers of the location of written training materials and shall make these materials readily available, without cost to the affected workers.

(c) Heat-Stress Safety Data Sheet

(1) The information specified in (a) of this section shall be recorded on a heat-stress safety data sheet or on a form specified by the Occupational Safety and Health Administration (OSHA).

(2) In addition, the safety data sheet shall contain:

(i) Emergency and first aid procedures, and

(ii) Notes to physician regarding classification, medical aspects, and prevention of heat injury and illness. These notes shall include information on the category and clinical features of each injury and illness, predisposing factors, underlying physiologic disturbance, treatment, and prevention procedures (see Table IV-1).

Section 7 - Control of Heat Stress

(a) General Requirements

(1) Where engineering and work practice controls are not sufficient to reduce exposures to or below the applicable RAL or REL, they shall, nonetheless, be used to reduce exposures to the lowest level achievable by these controls and shall be supplemented by the use of heat-protective clothing or equipment, and a heat-alert program shall be implemented as specified in (d) of this section.

(2) The employer shall establish and implement a written program to reduce exposures to or below the applicable RAL or REL by means of engineering and work practice controls.

(b) Engineering Controls

(1) The type and extent of engineering controls required to bring the environmental heat below the applicable RAL or REL can be calculated using the basic heat exchange formulae (e.g., Chapters III and VI). When the environmental heat exceeds the applicable RAL or REL, the following control requirements shall be used.

(a) When the air temperature exceeds the skin temperature, convective heat gain shall be reduced by decreasing air temperature and/or decreasing the air velocity if it exceeds 1.5 meters per second (m/sec) (300 ft/min). When air temperature is lower than skin temperature, convective heat loss shall be increased by increasing air velocity. The type, amount, and characteristics of clothing will influence heat exchange between the body and the environment.

(b) When the temperature of the surrounding solid objects exceeds skin temperature, radiative heat gain shall be reduced by: placing shielding or barriers, that are radiant-reflecting or heat-absorbing, between the heat source and the worker; by isolating the source of radiant heat; or by modifying the hot process or operation.

(c) When necessary, evaporative heat loss shall be increased by increasing air movement over the worker, by reducing the influx of moisture from steam leaks or from water on the workplace floors, or by reducing the water vapor content (humidity) of the air. The air and water vapor permeability of the clothing worn by the worker will influence the rate of heat exchange by evaporation.

(c) Work and Hygienic Practices

(1) Work modifications and hygienic practices shall be introduced to reduce both environmental and metabolic heat when engineering controls are not adequate or are not feasible. The most effective preventive work and hygienic practices for reducing heat stress include, but are not limited to the following parts of this section:

(a) Limiting the time the worker spends each day in the hot environment by decreasing exposure time in the hot environment and/or increasing recovery time spent in a cool environment;

(b) Reducing the metabolic demands of the job by such procedures as mechanization, use of special tools, or increase in the number of workers per task;

(c) Increasing heat tolerance by a heat acclimatization program and by increasing physical fitness;

(d) Training supervisors and workers to recognize early signs and symptoms of heat illnesses and to administer relevant first aid procedures;

(e) Implementing a buddy system in which workers are responsible for observing fellow workers for early signs and symptoms of heat intolerance such as weakness, unsteady gait, irritability, disorientation, changes in skin color, or general malaise; and

(f) Providing adequate amounts of cool, i.e., 10° to 15°C (50° to 59°F) potable water near the work area and encouraging all workers to drink a cup of water (about 150 to 200 mL (5 to 7 ounces) every 15 to 20 minutes. Individual, not communal, drinking cups shall be provided.

(d) Heat-Alert Program

A written Heat-Alert Program shall be developed and implemented whenever the National Weather Service or other competent weather forecast service forecasts that a heat wave is likely to occur the following day or days. A heat wave is indicated when daily maximum temperature exceeds 35°C (95°F) or when the daily maximum temperature exceeds 32°C (90°F) and is 5°C (9°F) or more above the maximum reached on the preceding days. The details for a Heat-Alert Program are described in Chapter VI, Section C.

Section 8 - Recordkeeping

(a) Environmental Surveillance

(1) The employer shall establish and maintain an accurate record of all measurements made to determine environmental and metabolic

heat exposures to workers as required in Section 1 of this recommended standard.

(2) Where the employer has determined that no metabolic heat measurements are required as specified in Section 1, Part (c)(2) of this recommended standard, the employer shall maintain a record of the screening estimates relied upon to reach the determination.

(b) Medical Surveillance

The employer shall establish and maintain an accurate record for each worker subject to medical surveillance as specified in Section 2 of this recommended standard.

(c) Surveillance of Heat-Induced Sentinel Health Events

The employer shall establish and maintain an accurate record of the data and analyses specified in Section 3 of this recommended standard.

(d) Heat-Induced Illness Surveillance

The employer shall establish and maintain an accurate record of any heat-induced illness or injury and the environmental and work conditions at the time of the illness or injury.

(e) Heat Stress Tolerance Augmentation

The employer shall establish and maintain an accurate record of all heat stress tolerance augmentation for workers by heat acclimatization procedures and/or physical fitness enhancement.

(f) Record Retention

In accordance with the requirements of 29 CFR 1910.20(d), the employer shall retain records described by this recommended standard for at least the following periods:

(1) Thirty years for environmental monitoring records,

(2) Duration of employment plus 30 years for medical surveillance records,

(3) Thirty years for surveillance records for heat-induced SHE's, and

(4) Thirty years for records of heat stress tolerance augmentation

(g) Availability of Records

(1) The employer shall make worker environmental surveillance records available upon request for examination and copying to the subject worker or former worker or to anyone having the specific

written consent of the subject worker or former worker in accordance with 29 CFR 1910.20.

(2) Any worker's medical surveillance records, surveillance records for heat-induced SHE's, or records of heat stress tolerance augmentation that are required by this recommended standard shall be provided upon request for examination and copying to the subject worker or former worker or to anyone having the specific written consent of the subject worker or former worker.

(h) Transfer of Records

(1) The employer shall comply with the requirements on the transfer of records set forth in the standard, Access to Medical Records, 29 CFR 1910.20(h).

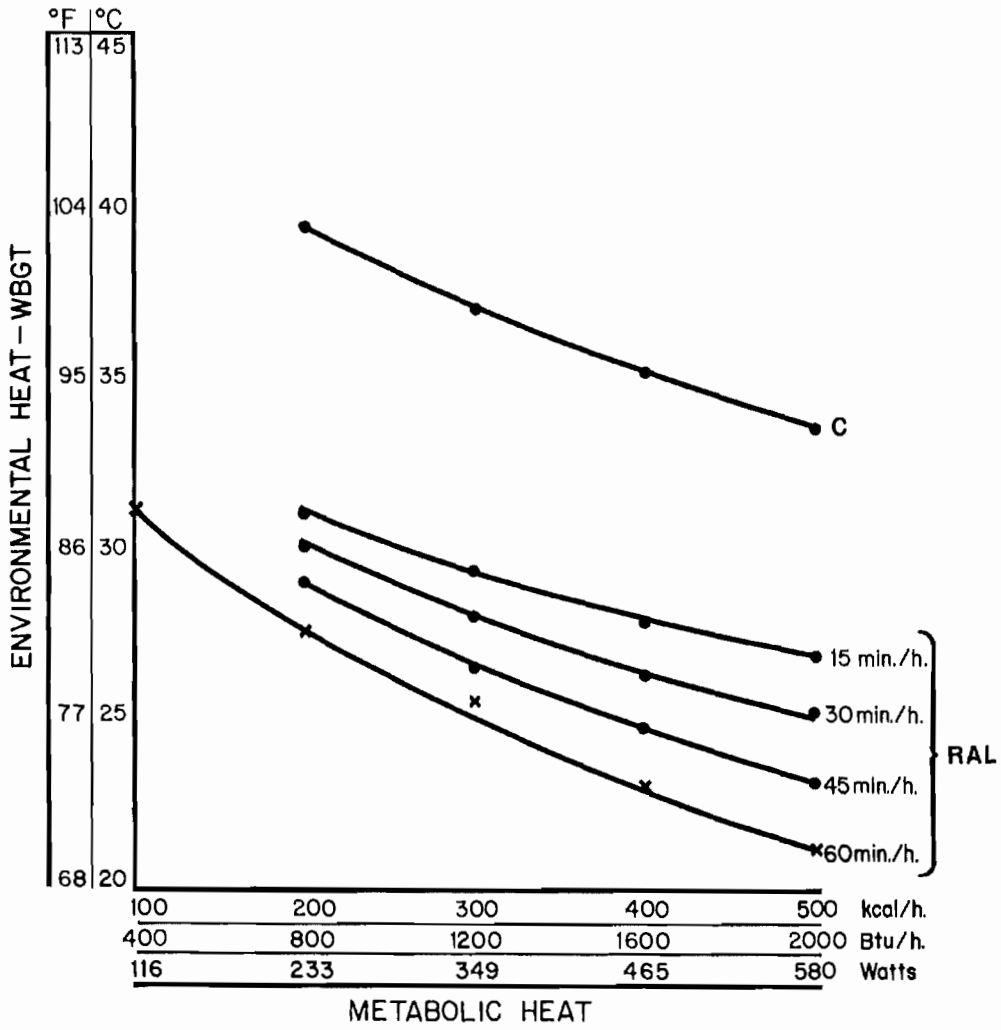

Figure 1. Recommended Heat-Stress Alert Limits
Heat-Unacclimatized Workers

C = Ceiling Limit
RAL = Recommended Alert Limit
*For "standard worker" of 70 kg (154 lbs) body weight and 1.8 m^2 (19.4 ft^2) body surface.
Based on References 2,3,4,5,6,7,8.

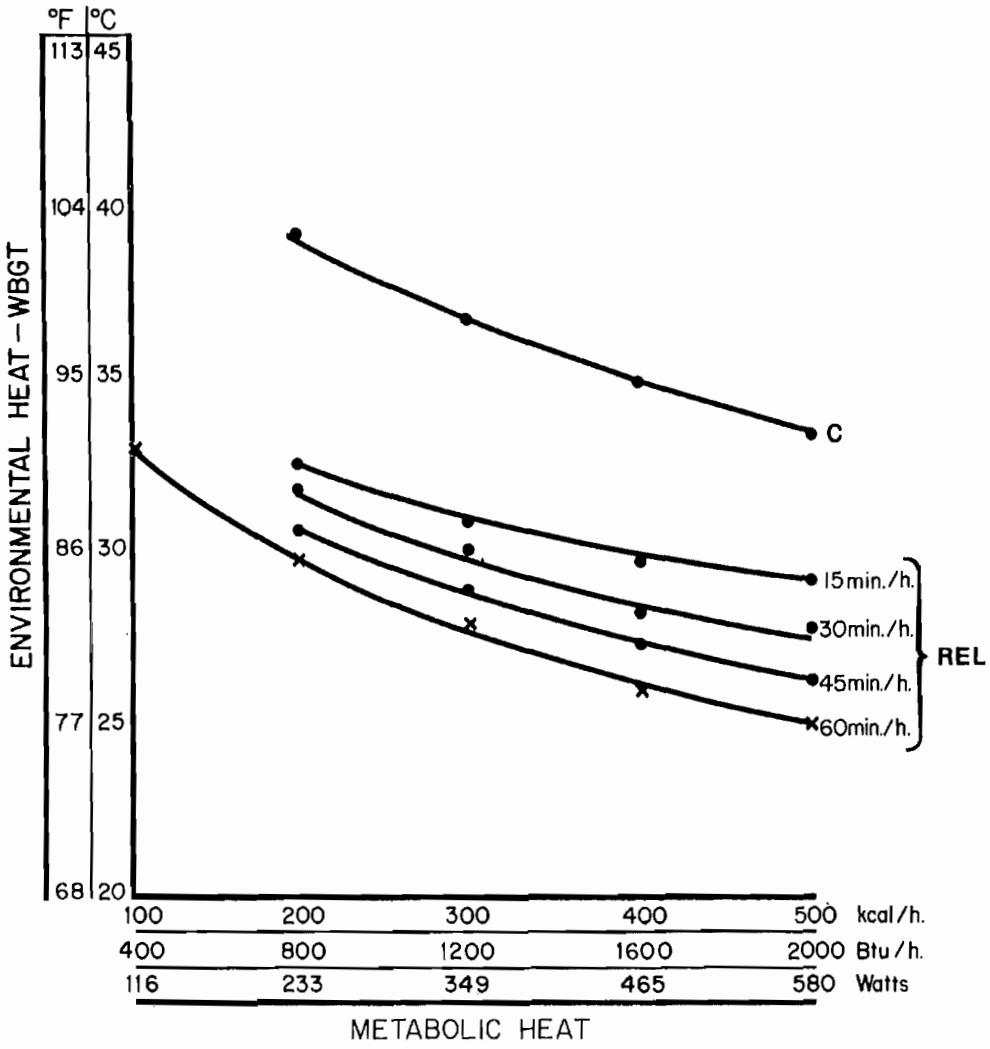

Figure 2. Recommended Heat-Stress Exposure Limits
Heat-Acclimatized Workers

C = Ceiling Limit
REL = Recommended Exposure Limit
*For "standard worker" of 70 kg (154 lbs) body weight and 1.8 m^2 (19.4 ft^2) body surface.
Based on References 2,3,4,5,6,7,8.

II. INTRODUCTION

Criteria documents are developed by the National Institute for Occupational Safety and Health (NIOSH), in response to section 20(a)(3) of the Occupational Safety and Health Act of 1970. In the Act, NIOSH is charged with developing criteria documents for toxic chemical substances and harmful physical agents which will describe exposure levels that are safe for various periods of employment including but not limited to the exposure levels at which no worker will suffer impaired health or functional capacities or diminished life expectancy as a result of any work experience. Environmental heat is a potentially harmful physical agent. This document presents the criteria and recommendations for a standard that were prepared to meet the need for preventing heat-induced health impairment resulting from exposure to occupational heat stress.

This document is an update of the Criteria for a Recommended Standard.... Occupational Exposure to Hot Environments (HSM-10269) published by NIOSH in January 1972 [9]. In June 1972, NIOSH sent the criteria document to the Occupational Safety and Health Administration (OSHA). In January 1973, the Assistant Secretary of Labor for Occupational Safety and Health appointed a 15 member Standards Advisory Committee on Heat Stress to review the NIOSH criteria document and develop a proposed standard. The committee submitted a proposed standard to the Assistant Secretary of Labor, OSHA, in January 1974 [7]. A standard on occupational exposure to hot environments was not promulgated. The updating of this document is based on the relevant scientific data and industry experience that have accrued since the original document was prepared. The document presents the criteria, techniques, and procedures for the assessment, evaluation, and control of occupational heat stress by engineering and preventive work practices and those for the recognition, treatment, and prevention of heat-induced illnesses and unsafe acts by medical supervision, hygienic practices, and training programs.

The recommended criteria were developed to ensure that adherence to them will (1) protect against the risk of heat-induced illnesses and unsafe acts, (2) be achievable by techniques that are valid, reproducible, and available, and (3) be attainable by existing techniques. This recommended standard is also designed to prevent possible harmful effects from interactions between heat and toxic chemical and physical agents. The recommended environmental limits for various intensities of physical work as indicated in Figures 1 and 2 are not upper tolerance limits for heat exposure for all workers but rather levels at which engineering controls, preventive work and hygienic practices, and administrative or other control procedures should be implemented in order to reduce the risk of heat illnesses even in the least heat-tolerant workers.

Estimates of the number of industrial workers who are exposed to heat stress on the job are at best rough guesses. A review of the Statistical Abstracts of the United States, 105th edition 1985, for the number of workers in industries where heat stress is a potential safety and health hazard indicates that a conservative estimate would be 5 to 10 million workers [10].

A glossary of terms, symbols, abbreviations, and units of measure used in this document is presented in XII-A.

Municipal Heat Wave Response Plans

Susan M. Bernard, JD, DrPH, MPH, and Michael A. McGeehin, PhD, MSPH

Approximately 400 people die from extreme heat each year in the United States, and the risk of heat waves may increase as a result of global climate change. Despite the risk of heat-related morbidity and mortality, many cities lack written heat response plans. In a review of plans from 18 cities at risk for heat-related mortality, we found that many cities had inadequate or no heat response plans. This is an important area for further investigation and government attention. (*Am J Public Health.* 2004;94:1520–1522)

Heat-related mortality has historically been an important, yet underestimated, public health problem in the United States.[1–23] Since 1998, heat waves have resulted in more weather-related fatalities annually than any other natural disaster (about 400 deaths per year).[24] Heat wave effects may increase in the near future as a result of global climate change.[3,25–31]

People most at risk include elderly and very young persons, chronically ill patients, socially isolated people, urban residents, and people without access to air conditioning.[2,9,12,19,23,32–38] The public health effect of heat waves is associated with factors such as the intensity, duration, time of year, and extent to which the heat event deviates from the norm experienced by the population.[2,39–48]

As with other disasters, municipalities must be prepared for heat waves.[2,21,49] However, many at-risk cities have minimal or no heat wave response plans. In the summer of 2002, we reviewed response plans from 18 cities with a history of, or at risk for, heat-related mortality. Our goal was to determine whether these plans reflected awareness of the risk, risk factors, and response measures. What we found suggests that this is an important area for future investigation and government attention.

One third of the cities we contacted lacked any written heat planning (including heat-specific measures incorporated into all-hazards plans). Although 10 cities did have stand-alone heat response plans, almost one third of these were cursory.

Heat wave planning should be organized around a few central principles: (1) identification of a lead agency and participating organizations; (2) use of a consistent, standardized warning system activated and deactivated according to weather conditions; (3) use of communication and public education; (4) implementation of response activities targeting high-risk populations; (5) collection and evaluation of information; and (6) revision of the plan.[5,49] Effective planning may have played a role in reducing heat-related mortality during recent heat waves.[38,50,51]

Comprehensive heat response requires involvement of many city departments and nongovernmental organizations. In most of the reviewed plans, heat response was coordinated by public safety or emergency management offices, but a few were based in the health department. Some plans identified other participating organizations.

Response measures should be implemented as appropriate on the basis of local data showing weather thresholds associated with increased mortality.[31,52] Plans we reviewed initiated response on the basis of criteria, including threshold temperature; heat index, which incorporates heat and humidity; and a synoptic air mass method.[53–60] Regardless of the method used to identify a threshold weather event for response, heatstroke has a fast onset and poor survival rate,[61] so prevention efforts must begin when high temperatures are forecast rather than when they arrive.[16,21] A few plans took a phased approach to response, which is valuable because mortality increases nonlinearly with the duration of the heat event.[2,8,14,39,60,62]

Plans also should be clear regarding when deactivation is appropriate. During the 1999 Midwest heat wave, a brief break in the heat triggered a declaration in Chicago, Ill, that the emergency was over, while in St. Louis, Mo, heat response remained effective because of conservative deactivation criteria; this early declaration may have led to additional deaths in Chicago.[62]

In general, the plans detailed public information procedures. However, most provided for public outreach only when a heat wave was imminent or had already begun.

Because access to air conditioning is the most effective intervention to reduce mortality from heat waves,[2,34] many cities took steps to increase such access by opening buildings to the public. Unfortunately, centralized cooling centers have not proved effective in reaching the most at-risk seniors.[38,62]

Targeted outreach is critical to reaching the socially isolated.[1,50,63] Two cities collected voluntarily submitted names of at-risk individuals to be contacted during a heat emergency, but it was unclear how well this service operated. Four cities asked neighborhood organizations or mail or utility workers to check on at-risk individuals. Several cities publicly urged people to check in with elderly individuals; the usefulness of this advice is unclear.[38] Although people with mental or chronic illness form a significant proportion of the victims of recent heat waves,[38,64] only 1 plan emphasized reaching out to disabled persons. Only 2 plans addressed the shelter and water needs of the homeless.[52]

Five of the cities reported fan distribution programs, despite evidence that fans do not reduce mortality risk during heat waves and can increase heat stress if used improperly.[2,14,36,38,65] In 3 cities, residential water service cannot be shut off during extreme heat events, and several cities took steps to prevent illegal use of fire hydrants.

Surveillance on weather, hospital and ambulance use, medical examiner reports,[23,35] electric and water supply and demand, and outreach efforts—critical to determining the

effectiveness of interventions—was absent or minimal under most plans reviewed.

Comprehensive response plans are necessary to reduce heat-related morbidity and mortality. Plans should provide for coordinated action across government authorities, involve private sector participants, and be responsive to variable risk factors. More research is needed on plan effectiveness. ■

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Contributors

S.M. Bernard conducted the investigation and was primarily responsible for the analysis and writing of the brief. M.A. McGeehin identified the need for such an investigation and assisted with the analysis and background.

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bly also MDR HIV. The findings in this report, along with increasing syphilis rates, continuing gonorrhea transmission, and the emergence of lymphogranuloma venereum in HIV-positive MSM, reflects a resurgence of unsafe sex among MSM. This behavior also has been associated with increasing use of methamphetamine (*7*).

The genotype data collected by NYCDOH indicated a low prevalence of MDR genotypes among persons who had not been treated with ARVs and who had HIV infections diagnosed during June 1, 2004–June 30, 2005. Drug-resistant HIV compromises the effectiveness of standard ARV regimens and can limit the treatment options available to persons with newly diagnosed HIV infection (*6*). Therefore, CDC has provided funding to four city and 17 state health departments to conduct drug-resistance surveillance on remnant sera obtained from all patients with newly diagnosed HIV infection (*8*). Provisional data from these areas indicate that as many as 15% of these patients are infected with an HIV strain that has mutations associated with resistance to ARVs, and 3.2% have mutations associated with resistance to two or more classes of such medications.[§]

Case reports such as the one described here and results from surveillance of newly diagnosed, drug-resistant HIV infections contributed to recent changes in HIV-1 treatment guidelines issued by the U.S. Department of Health and Human Services (*9*). These guidelines now recommend performing drug-resistance testing before initiation of therapy in patients who have never received ARV treatment. To reduce HIV-associated morbidity and mortality in the United States, public health officials should intensify measures to improve early diagnosis, partner notification, and prevention counseling for persons (particularly MSM) who are HIV positive and should conduct population-based genotype surveillance to monitor the emergence of unusual strains of HIV, particularly those with mutations associated with ARV resistance (*8,10*).

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Heat-Related Deaths — United States, 1999–2003

Heat-related illnesses (e.g., heat cramps, heat exhaustion, heat syncope, or heatstroke) can occur when high ambient temperatures overcome the body's natural ability to dissipate heat (*1*). Older adults, young children, and persons with chronic medical conditions are particularly susceptible to these illnesses and are at high risk for heat-related mortality (*2*). Previous analyses of the risk factors associated with heat-related deaths* have been based on the underlying cause† entered on the death certificate (*4,5*) and have not included decedents for whom hyperthermia was listed as a contributing factor but not the underlying cause of death. This report describes an analysis in which number of heat-related deaths were counted, including deaths in which hyperthermia was listed as a contributing factor on the death certificate. The analysis revealed that including these deaths increased the number of heat-related deaths by 54% and suggested that the number of heat-related deaths is underestimated.

CDC uses information from death certificates categorized by codes from the *International Classification of Diseases* to estimate national mortality trends. These data, collected and submitted by states, were used to determine the number of deaths in the United States during 1999–2003 that had expo-

* Defined as a death in which exposure to high ambient temperatures either caused the death or contributed to it substantially, the decedent had a body temperature at the time of collapse >105°F (>40.6°C), the decedent had a history of exposure to high ambient temperature, and other causes of hyperthermia could reasonably be excluded (*3*).

† The underlying cause of death is defined as the disease or injury that initiated the chain of events that lead directly and inevitably to death. Contributing conditions, or factors, are defined as diseases, injuries, or complications that directly caused the death. A sample death certificate, showing underlying and contributing causes of death, is available at http://www.cdc.gov/nchs/data/dvs/death11-03final-acc.pdf.

sure to excessive natural heat[§] recorded as the underlying cause (code X30 from ICD, tenth revision [ICD-10]), hyperthermia[¶] recorded as a contributing factor (ICD-10 code T67) (*6*), or both.

During 1999–2003, a total of 3,442 deaths resulting from exposure to extreme heat were reported (annual mean: 688). For 2,239 (65%) of these deaths, the underlying cause of death was recorded as exposure to excessive heat; for the remaining 1,203 (35%), hyperthermia was recorded as a contributing factor. Deaths among males accounted for 66% of deaths and outnumbered deaths among females in all age groups (Figure). Of the 3,401 decedents for whom age information was available, 228 (7%) were aged <15 years, 1,810 (53%) were aged 15–64 years, and 1,363 (40%) were aged ≥65 years. The state with the highest average annual hyperthermia-related death rate during 1999–2003 was Arizona (1.7 deaths per 100,000 population), followed by Nevada (0.8) and Missouri (0.6).

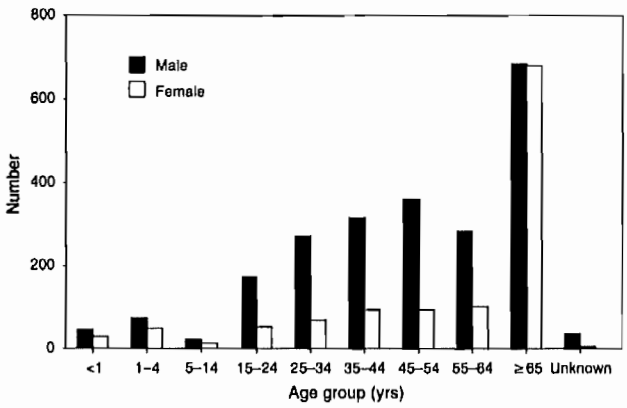
Cardiovascular disease was recorded as the underlying cause of death in 681 (57%) of cases in which hyperthermia was a contributing factor (Table). Approximately 70% of these heat-related cardiovascular deaths occurred among persons with reported chronic ischemic heart disease. External causes (e.g., unintentional poisonings) were documented as the underlying cause of 345 (29%) deaths in which hyperthermia was a contributing factor. Endocrine, nutritional, and metabolic disorders (e.g., diabetes mellitus) were the underlying causes in 38 (3%) of total deaths. All other underlying causes, including infection and psychiatric disorders, accounted for 139 (11%) deaths.

TABLE. Selected underlying causes of death with hyperthermia* as a contributing factor† — United States, 1999–2003

Underlying cause of death	No.	(%)
Cardiovascular diseases	**681**	**(56.6)**
Chronic ischemic heart disease	473	(39.3)
Acute ischemic heart disease	63	(5.2)
Hypertensive heart disease without congestive heart failure	60	(5.0)
Other cardiovascular diseases	85	(7.1)
External causes of morbidity and mortality	**345**	**(28.7)**
Accidental poisoning by and exposure to noxious substances	51	(4.2)
Assault	63	(5.2)
Other external causes of morbidity and mortality	231	(19.2)
Diseases of the respiratory system	**37**	**(3.1)**
Chronic obstructive pulmonary disease, unspecified	27	(2.2)
Other diseases of the respiratory system	10	(0.8)
Endocrine, nutritional, and metabolic disorders	**38**	**(3.2)**
Unspecified diabetes mellitus	26	(2.2)
Other endocrine, nutritional, and metabolic disorders	12	(1.0)
Mental and behavioral disorders	**29**	**(2.4)**
Mental and behavioral disorders due to alcoholism	21	(1.7)
Other mental and behavioral disorders	8	(0.7)
Diseases of the digestive system	**22**	**(1.8)**
Fibrosis and cirrhosis of the liver	15	(1.2)
Other diseases of the digestive system	7	(0.6)
Other diseases of the nervous, infectious, immune, and genitourinary systems and neoplasms	**51**	**(4.2)**

* Abnormally high body temperature caused by the body's inability to dissipate heat.
† N = 1,203.

FIGURE. Number of heat-related deaths,* by sex and age group — United States, 1999–2003



* Exposure to extreme heat is reported as the underlying cause of or a contributing factor to death (N = 3,442).

§ Heat-related deaths can also be caused by exposure to excessive heat of man-made origin (e.g., from saunas or furnace malfunctions; *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision* [ICD-10] code W92) and can include homicides and suicides involving exposure to excessive heat. Deaths from these causes were not included in this analysis.
¶ Abnormally high body temperature caused by the body's inability to dissipate heat.

Reported by: *GE Luber, PhD, CA Sanchez, MD, Div of Environmental Hazards and Health Effects, National Center for Environmental Health/Agency for Toxic Substances and Disease Registry (proposed); LM Conklin, MD, EIS Officer, CDC.*

Editorial Note: In this analysis, the inclusion of hyperthermia as a contributing cause of death increased by 54% the total number of heat-related deaths during 1999–2003 that would have been counted through inclusion of a heat-related underlying cause alone. Because heat-related illnesses can exacerbate existing medical conditions and death from heat exposure can be preceded by various symptoms, heat-related deaths can be difficult to identify when illness onset or death is not witnessed by a clinician. In addition, the criteria used to determine heat-related causes of death vary among states. This can lead to underreporting heat-related deaths or to reporting heat as a factor contributing to death rather than the underlying cause (*3*). The demographics (e.g., sex, age group, and state) of the decedents described in this report are

consistent with previous descriptions of persons at risk for heat-related deaths (*4,5*).

This analysis also provides additional information on the underlying causes of death in which hyperthermia was a contributing factor. Although this report might still underestimate the extent of overall heat-related morbidity and mortality, the inclusion of hyperthermia as a contributing factor to death provides a more comprehensive view of the actual effects of heat-related illnesses. The association between cardiovascular disease and heat-related death is well established (*7*); this analysis suggests the need for additional investigations of the association between noncardiovascular conditions, such as endocrine and respiratory diseases, and the risk for heat-related death.

Continued exposure to excessive heat can lead to hyperthermia or death. Of the heat-related illnesses, heat exhaustion and heatstroke are the most serious. Heat exhaustion is characterized by muscle cramps, fatigue, headache, nausea or vomiting, and dizziness or fainting. The skin is often cool and moist, indicating that the body's mechanism for cooling itself (i.e., sweating) is still functioning. The pulse rate is typically fast and weak, and breathing is rapid and shallow. If untreated, heat exhaustion can progress to heatstroke (*1*). Heatstroke is a serious, life-threatening condition characterized by a high body temperature (>103°F [>39.4°C]); red, hot, and dry skin (no sweating); rapid, strong pulse; throbbing headache; dizziness; nausea; confusion; and unconsciousness. Symptoms can progress to encephalopathy, liver and kidney failure, coagulopathy, and multiple organ system dysfunction (*2*). Prompt treatment of heat-related illnesses with aggressive fluid replacement and cooling of core body temperature is critical to reducing morbidity and mortality (*2*).

Many heat-related deaths, regardless of whether they are associated with chronic medical conditions, are preventable. During periods of extreme heat, heat-related illnesses can be prevented by avoiding strenuous outdoor activities, drinking adequate amounts of fluid, avoiding alcohol consumption, wearing lightweight clothing, and using air-conditioning. Groups at high risk include young children, persons aged >65 years, persons who do strenuous activities outdoors, and persons with chronic (particularly cardiovascular) medical conditions (*8*).

During heat waves, young children, older adults, and chronically ill persons should be checked frequently by relatives, neighbors, and caretakers to evaluate their heat exposure, recognize symptoms of heat-related illness, and take appropriate preventive action. Regardless of the outdoor temperature, parents and other child-care providers should never leave children alone in cars and should ensure that children cannot lock themselves inside enclosed spaces, such as the trunks of automobiles.

Communities can prepare for heat-related illnesses by creating well-defined heat response plans (HRPs) (*9*). Both governmental and nongovernmental organizations, each with specific roles and responsibilities, can be involved in this planning. HRP protocols and communication tools should be reviewed annually, before the summer months begin. The HRPs should identify populations at high risk for heat-related illness and death and determine which strategies will be used to reach them during heat emergencies. The HRP should also include specific criteria for activation and deactivation of the plan. Postemergency evaluations of HRPs are necessary to make appropriate revisions and improve plan effectiveness.

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Chagas Disease After Organ Transplantation — Los Angeles, California, 2006

Chagas disease is an infection caused by the parasite *Trypanosoma cruzi*. Reduviids (i.e., "kissing bugs") transmit the parasite through infected feces. *T. cruzi* also can be transmitted congenitally and through blood transfusion or organ transplantation. The infection is lifelong if left untreated; the majority of infected persons are asymptomatic, and their disease remains undiagnosed. Although routine serologic testing of organ and blood donors is performed in areas of Latin

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Commentary

A critical comment on heat wave response plans

Julio Díaz[1], Cristina Linares[1], Aurelio Tobías[2]

The *European Journal of Public Health*,[1] among others,[2,3] has recently reported that heat waves are one of the main health risks stemming from climate change. Environmental studies have shown that heat waves will become more intense, more frequent, and longer in the near future.[4] The way to deal with this risk is a city heat wave early warning system. In this regard, we like to remark a significant point to which most heat wave response plans fail to give proper consideration.

Most warning systems developed in European cities are activated when ambient temperature exceeds an established threshold. Nevertheless, this threshold is usually based on the relationship between mortality and temperature, to the detriment of other health outcomes, such as hospital admissions. It has recently been shown that mortality and hospital-admission patterns are different during heat waves. In the city of Madrid, the main difference between mortality and hospital-admission patterns has been reported to be due to heat-related disease causes,[5] meaning that, when there is a heat wave, circulatory diseases are the main cause of mortality, though not of hospital admission. These results agree with those previously published on the short-term effects of heat waves in Greater London.[6] High temperatures provoke increased platelet and red cell counts, blood viscosity, and plasma cholesterol levels during heat stress, and mortality from coronary and cerebral thrombosis.[7] Within a short time of exposure to high temperatures, affected subjects rapidly progress to fatal health outcomes.[8] Accordingly, such persons die before they can be admitted to hospital, a factor that is of the essence when it comes to designing heat wave warning systems. Heat wave response plans should therefore be triggered before the arrival of the heat wave, and this can be easily achieved thanks to the fact that meteorological forecasting is highly reliable within a 24–48 h timeframe. Early activation of prevention plans, particularly insofar as social services are concerned, allows for early observation of persons susceptible to being affected by heat waves and implementation of actions before onset of the first symptoms can lead to premature death due to excess heat.

In conclusion, prevention of adverse heat wave-related health effects must be set in motion 1 or 2 days before and *not* on the same day as the designated mortality–threshold temperature is exceeded.

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Temperature and cardiovascular mortality.

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AUTHOR: Khaw, Kay-Tee.

SECTION: Commentary

LENGTH: 1025 words

Each winter, deaths in most countries are 10-25% higher than in the rest of the year n 1 In Britain, winter brings an estimated 40 000 excess deaths. Most of these deaths are due to cardiovascular disease, predominantly heart attacks and strokes. What brings about the winter excess mortality? Seasonal changes in environmental and other factors such as air pollution, sunlight exposure, influenza incidence, and diet have variously been implicated; of these, temperature is a leading candidate n 1-5

Pan and colleagues now report a striking U-shaped relation between outdoor temperature and cardiovascular disease mortality rates in Taiwan, with a smooth downward gradient between 10 and 29 degrees C and a sharp upswing thereafter at about 32 degrees C. These findings are especially interesting since Taiwan is a subtropical country, with a wide temperature range and patterns of mortality that differ from those in northern Europe or North America. In particular, stroke is more common than ischaemic heart disease, haemorrhagic stroke being more common than thrombotic stroke, a reversal of the patterns seen in most western countries.

Is the association causal or is temperature a surrogate measure for related factors such as air quality or sunlight exposure? The consistency of the association in different countries and communities, over time, and in different age groups, despite the large measurement errors that are unavoidable with routinely collected statistical data, is convincing. The close day-to-day association suggests a surprisingly dynamic relationship. Clinical and laboratory data indicate several biologically plausible mechanisms, including effects of changing temperature on haemostasis, blood viscosity, lipids, the sympathetic nervous system, and vasoconstriction n 6-8 There is also a strong inverse relation between blood pressure and temperature n 9 These factors may interact. Thus, raised blood pressure can precipitate myocardial ischaemia by increasing cardiac work, while concurrent peripheral vasoconstriction results in greater mechanical vessel stress and vascular damage.

The next question relates to the nature of the exposure-eg, duration, timing, dynamic changes, and, in particular, whether outdoor or indoor temperature exposure is more important. Although outdoor temperatures have been used in most mortality analyses, outdoor temperature may also be a marker for indoor temperature exposure since they are closely related: despite heating, mean temperatures are colder indoors in winter than in summer. Some observations indicate that indoor temperatures have an impact. The magnitude of the winter excess mortality is only weakly related to latitude: Scandinavian countries (which tend to have indoor heating and building insulation) have less seasonal

variation in mortality than more southern countries such as Portugal and Israel n 1 where central heating is not widespread and where indoor temperature may vary more in line with outdoor temperature throughout the year. The gradient of the inverse relation of mortality with outdoor temperature is much steeper in areas where heating is less widespread, even though they may have warmer winters-eg, Los Angeles has a steeper gradient than New York n 5 Ambient indoor temperature has a direct influence on blood pressure, and one study showed that the seasonal variation in blood pressure related independently to both indoor and outdoor temperature, indoor temperature having the stronger impact. A 4 degrees C decrease in living room temperature was associated with a 5 mm Hg rise in systolic blood pressure, equivalent to about 5% increase in cardiovascular risk n 9 The social class gradient observed in Britain-the magnitude of the excess winter mortality in men in social class V is twice that of men in social class I-accords with different housing patterns and heating n 1

The magnitude of the association and optimum range are notable. The temperature with lowest cardiovascular disease mortality (29 degrees C) is higher in Taiwan than in the Netherlands (17 degrees C, for all-cause mortality). Kunst n 4 found that, in the Netherlands, most cold-related deaths were cardiovascular while most heat-related deaths were respiratory. It would be interesting to know whether all- cause mortality in Taiwan shows the same pattern as cardiovascular disease mortality. The differences may reflect the fact that there are not many days above 18 degrees C days in the Netherlands, home heating patterns (low outdoor temperature in the Netherlands may be associated with warmer indoor temperatures than in Taiwan), or acclimatisation effects. Studies from the USA n 3 and elsewhere n 5 show a similar U-shape, with optimum mean outdoor temperatures for coronary and stroke mortality around 25-27 degrees C.

Whilst high temperatures (>30 degrees C) seem adverse, the number of days with mean temperatures as high as that is very small in most countries. By contrast, many countries experience daily temperatures over the range where there is a continuous smooth inverse association with mortality. In a British study, maximum mean indoor and outdoor temperatures recorded over a year did not exceed 30 degrees C; means over the year were much lower (2-16 degrees C outdoors, 21-24 degrees C indoors) n 9 Kunst n 4 estimated that a drop of 3 degrees C within the outdoor range - 15 degrees C to 17 degrees C was associated with a 1% mortality increase. The magnitude of effect for cardiovascular disease in Taiwan was much greater-around 3-10% for every drop of 3 degrees C within the range 11-29 degrees C. An analysis by Boardman n 10 of the relation between average winter indoor temperature and winter excess mortality in various countries showed a 1% mortality decline for every 1 degrees C increase in temperature over the range 14-21 degrees C. If changing indoor as well as outdoor temperature influences mortality- and the evidence increasingly supports this view-the implications are considerable. Social and economic policies that support energy- efficient housing and central heating may also be good for health.

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Killing Heat

HEATSTROKE is common and often deadly. The pathophysiology of the syndrome is understood, and effective treatment has been well defined.[1-3] Clinicians know that patients' chances of survival depend on prompt diagnosis and rapid cooling. Otherwise, a cascade of events will lead to irreversible injury or death.

Heatstroke is preventable. We know the epidemiologic features of the disorder, and effective steps to prevent heatstroke have been clearly defined.[4-10] To avoid heat-related deaths, a community must recognize the impending problem promptly. Once a heat-related emergency is identified, effective measures must be implemented at once. Otherwise, many deaths will follow.

In this issue of the *Journal,* researchers from the Centers for Disease Control and Prevention and the Illinois and Chicago departments of public health report a case–control study of deaths during the Chicago heat wave of 1995.[11] As in other heat waves, the vast majority of victims were elderly inner-city residents.[4-9] By comparing a stratified sample of those who died with randomly selected controls matched with them according to age and neighborhood of residence, the authors show that the people who died of heat-related causes were more likely than controls to have preexisting medical problems such as mental illness, pulmonary disease, or heart disease.

The effect of social isolation was even more striking. Living alone or being confined to bed was associated with a marked increase in the risk of heat-related death, even among those who had regular contact with a visiting nurse or another social-service agency. Conversely, access to transportation was protective, as was having a working air conditioner or spending a few hours each day in an air-conditioned place.[10]

The most obvious implication of these finding is that opening shelters is not enough. Communities must make a determined effort to seek out the elderly, the infirm, and the shut-in. Aggressive efforts to reach out to the most vulnerable can markedly reduce the toll of heat-related deaths. It has been done before, and it works.

Sixteen years ago, heat claimed the lives of 88 people in Memphis, Tennessee. An analysis of morbidity and mortality during the heat wave revealed that heat-related visits to hospital emergency departments increased sharply three days before the first death.[4] A shelter was opened, but it was used by only a small fraction of those at risk.

In response to these observations, Memphis has adopted a more active approach to future heat waves (Edmiston PW, Division of Community Services, Shelby County: personal communication). At the beginning of summer, officials initiate sentinel surveillance at emergency departments in the area in order to detect any sudden increase in heat-related visits. A 24-hour telephone line is maintained for advice and instructions. When the temperature begins to rise, the news media print or broadcast stories about the importance of increasing fluid intake, reducing physical activity, and seeking medical care for heat-related problems. Citizens with air conditioning are urged to check on elderly relatives, friends, and neighbors and to take them in as guests. Health Department inspectors visit nursing homes and personal care homes (where residents receive assistance but not skilled nursing) to check for excessive temperatures, and the county's Office on Aging contacts clients who have had problems in the past. Those who cannot cope with the heat are given a screening medical examination, then transported to air-conditioned shelters.

At the first sign of an increase in heat-related visits to emergency departments, efforts to reach people at high risk are intensified. Candidates for the shelter are actively sought out by visiting nurses, Meals on Wheels workers, and other service providers. Letter carriers play an important part by hand-delivering the mail to elderly customers on their routes. Although programs like this one have not been formally evaluated, Shelby County has recorded no more than 11 heat-related deaths in any summer since 1980, and the median number of heat-related deaths has been only 2 per year.

Can communities do more? Absolutely. Long-standing plans should be revised to incorporate new findings. Many agencies give electric fans to poor citizens to help them cope with the heat, but fans are useless when heat and humidity reach dangerous levels.[6,8-11] Money spent on fans should be redirected elsewhere. Heatstroke can develop quickly in previously asymptomatic persons.[5] Therefore, patients at high risk should be urged to move to cooler quarters, even if they appear to be coping with the heat. Since access to air conditioning for even a few hours each day is protective, local governments in high-risk areas of the country should consider modifying their

building codes to require every hotel and apartment building to have an air-conditioned lobby.[6,7,11]

Unfortunately, well-intentioned efforts can be hampered by suspicion and fear.[4] Air conditioners have been turned off by residents who thought they could not afford the utility bills and who later died of heat-related causes.[4,9] Windows that are nailed shut to thwart burglars lead to superheated homes. Some residents refuse to leave their homes unguarded. Others are simply too proud or too fearful to seek refuge in a public shelter.

New approaches are needed to address these fears. Relatives, friends, and neighbors can accommodate more people than the largest public shelter. Churches, synagogues, and mosques can help by reinforcing key safety messages. They should also open their facilities during the hottest hours of the day. Utility companies often suspend all shut-offs during heat waves, and debt-forgiveness programs can help with the bills of the truly needy.

If measures like these are taken, they should be widely publicized. Semenza and colleagues found that listening to the radio or reading a newspaper was associated with greater awareness of the health risk related to heat.[11] However, nearly everyone watches television. In a heat emergency, public education programs should rely heavily on television to get the message to all who need to hear it.

Every summer, officials issue warnings about the risk of heat-related illness.[8,9] Every summer, heat-related deaths occur anyway. The question is not how to prevent these tragedies, but why we do not do a better job. It is a sad fact that a large number of deaths at a single time and place evokes more powerful emotions than a much larger number of deaths spread more widely. The attention given to the recent crash of an airliner in Florida is in sharp contrast to the national response to the 1995 heat wave. Both were terrible events. One prompted an immediate "bottom-up" review of industry safety standards. The other was forgotten as soon as the temperature fell.

Few of the world's population centers can afford to ignore the potential dangers of a sustained heat wave. Every community should assess its resources and develop a contingency plan. Although heatstroke is amenable to medical treatment, control can best be achieved by applying the principles of public health. Sentinel surveillance, public education, outreach to vulnerable persons, and enlistment of the help of the entire community can save lives. The Chicago heat wave of a year ago should provide a lesson to us all.

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Clinical Implications of Basic Research

Molecular Mechanisms of Cocaine Addiction

EFFECTIVE medication for the treatment of cocaine overdose and addiction is a major unmet need of worldwide importance. In contrast to heroin, for which there are effective medications to treat both overdose (naloxone) and addiction (methadone), there are no pharmacologic treatments for any aspect of cocaine addiction. Recent discoveries indicating that an anti-cocaine medication is actually within reach are cause for great optimism. This work is defining specific molecular targets against which such a medication might be directed.

A recent study by Giros et al.[1] affirms the central importance of the dopamine-reuptake transporter in the behavioral and biochemical action of cocaine and defines it as a site on which efforts to develop an anti-cocaine medication should be focused. The dopamine-reuptake transporter controls the levels of dopamine in the synapse by rapidly carrying the neurotransmitter back into nerve terminals after its release (Fig. 1). Cocaine, which binds strongly to the dopamine-reuptake transporter, is a classic blocker of such reuptake after normal neuronal activity. Because of this blocking effect, dopamine remains at high concentrations in the synapse and continues to affect adjacent neurons, producing the characteristic cocaine "high."

Giros and his colleagues produced a strain of mice in which the gene encoding the dopamine transporter was disabled. These knockout mice, with no functional dopamine-reuptake transporters, did not respond to cocaine either biochemically or behaviorally. This study demonstrates that the transporter is necessary for cocaine to produce its psychostimu-

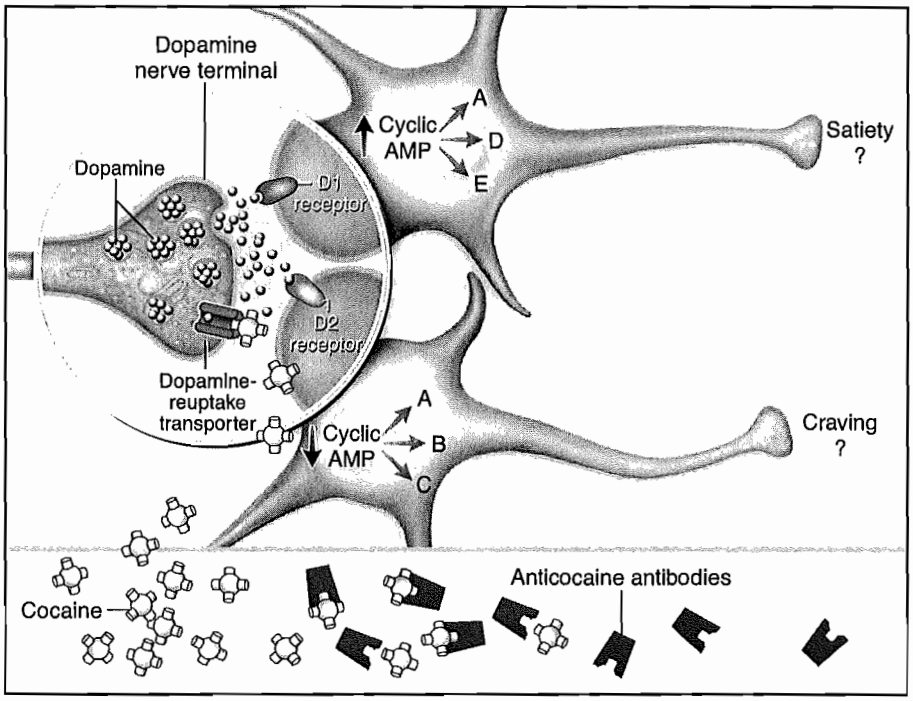

Figure 1. A Dopamine Nerve Terminal Innervating Neurons in the Nucleus Accumbens.

Three major sites of dopamine binding are shown: the D1 postsynaptic receptor, the D2 receptor, and the dopamine-reuptake transporter, which helps regulate synaptic levels of dopamine by carrying the transmitter back into the nerve terminal. Levels of dopamine at the synapse increase when cocaine inhibits the reuptake of dopamine by binding to the dopamine-reuptake transporter. In mice whose dopamine transporters have been eliminated by knockout of the gene for this molecule, synaptic dopamine levels are increased. Cocaine does not induce some biochemical or behavioral responses in these animals, because they have no functional dopamine transporters. The D1 receptors are mainly located on different neurons from the D2 receptors. D1 and D2 receptors have opposing intracellular and behavioral effects, probably mediated by multiple pathways (indicated here by letters A through E) and may differentially affect craving and satiety. Antibodies against cocaine appear to block the effects of the drug by keeping it from entering the central nervous system.

latory effects. Mice with the same deficiency are also being used to help answer questions about Parkinson's disease and certain psychiatric disorders that, like the effects of cocaine, are linked to a malfunction in the regulation of neurotransmission by dopamine.

Another advance in the identification of systems in the brain that relate to the addictive properties of cocaine was reported by Self et al.[2] There are multiple dopamine receptors on the postsynaptic membrane that respond differently to different compounds and are likely to serve different biologic functions (Fig. 1). Self et al. suggest that the D1 dopamine–receptor system is a target for medications to treat cocaine addiction. These investigators studied two compounds that stimulate either the D1 or the D2 dopamine receptor and tested the ability of the compounds to reinstate self-administration of cocaine in rats that had stopped seeking the drug. The rats were allowed to administer intravenous cocaine to themselves for two hours, after which saline was substituted for an equal period, during which self-administration progressively diminished or ceased. The researchers found that administering a D2 agonist led to a dramatic reinstatement of cocaine seeking, whereas a D1 agonist had virtually no effect. In a second test, in which rats were "primed" with cocaine, pretreatment with a D1 dopamine–receptor agonist suppressed cocaine seeking, whereas pretreatment with a D2 agonist increased it. That study clearly shows that D1- and D2-receptor agonists have opposite effects on cocaine-seeking behavior. More important, however, it suggests that D1-receptor agonists may diminish episodes of intense craving for cocaine in humans, preventing relapses in people who have stopped using the drug.

A third major discovery, reported by Carrera et al.,[3] demonstrates how immunologic techniques and a well-defined behavioral model can be successfully used in combination. These workers repeatedly injected rats over a 35-day period with a protein-conjugated analogue of cocaine that triggers the immune system to produce anti-cocaine antibodies. They then tested the animals' responses to injected cocaine. The immunized rats had significantly lower psychomotor responses to the stimulant effects of cocaine than control animals, showing that immunization against the behavioral effects of cocaine is possible. In addition, cocaine concentrations in the brains of immunized animals were 52 percent lower in striatal tissue and 77 percent lower in cerebellar tissue than those in the brains of controls, suggesting that anti-cocaine immunization prevents the uptake of cocaine into critical sites in the brain. This work suggests that it may be possible to vaccinate against the action of cocaine. Other researchers on the immunotherapy of drug abuse are exploring the use of antibodies and other external agents that affect the rate and direction of cocaine metabolism once the drug has been taken. A number of questions, including how long the immunization will remain effective and what risks are entailed in immunizing human subjects, need to be explored before any immunotherapy for cocaine addiction is ready for clinical trials.

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Editorials

ARTEMETHER IN SEVERE MALARIA — STILL TOO MANY DEATHS

THE impact of malaria is almost inconceivable to those who do not live in the tropics or subtropics. Each year, there are 300 million to 500 million new plasmodium infections and 1.5 million to 2.7 million deaths from malaria in the developing world. Since 1981 there have been an estimated 2.5 million deaths caused by infection with the human immunodeficiency virus worldwide, as compared with 20 million to 40 million deaths caused by malaria. Malaria is a major threat to travelers, and the U.S. military has had more casualties from malaria than from bullets in every campaign of the 20th century conducted in malarious regions.[1]

There is no licensed vaccine against malaria. Thus, efforts to reduce morbidity and mortality are limited to preventing contact between humans and the anopheles mosquitoes that transmit the infection, to chemoprophylaxis, and to the early identification and treatment of infected patients.

From the mid-17th century until the end of World War II, quinine was the primary treatment for malaria in most of the world. In the late 1940s, chloroquine replaced quinine, but by 1960 *Plasmodium falciparum* resistant to chloroquine was identified in Thailand and Colombia. Now there are few areas of the world without chloroquine-resistant *P. falciparum*. Although several drugs are available for treating uncomplicated malaria, in the past 15 to 20 years quinine has reemerged as the principal antimalarial agent for severe malaria, except in the United States, where quinidine (the dextrorotatory optical isomer of quinine) is used because of its widespread availability and similar efficacy.

Quinine is not an ideal antimalarial agent. It can cause toxic effects on the cardiovascular system, cinchonism, and painful local reactions after intramuscular administration, and because of its short half-life, the drug must be administered two to three times per day. This short half-life may explain the absence of widespread resistance to the drug, but the sensitivity of *P. falciparum* to quinine is declining and the clinical response to the drug is slowing in Thailand.[2] These developments have led to concern that in many places there will soon be no adequate treatment for severe malaria.

In the early 1970s, Chinese scientists identified artemisinin, a sesquiterpene lactone peroxide, as the principal active component of the traditional Chinese malaria remedy qinghaosu. Artemisinin and two derivatives, artesunate and artemether, are effective against multidrug-resistant *P. falciparum* and clear sensitive parasites from the blood more rapidly than other antimalarial agents.[3] Several studies in Southeast Asia have suggested that artemether is more effective than quinine in reducing the case fatality rates in severe malaria. In one of these studies in Thailand, the case fatality rate among quinine-treated patients was 37 percent[4] in 1995 in the same hospital where it had been 17 percent in the early 1980s.[5] This change suggested that the lower mortality associated with artemether was due not to its intrinsic superiority but to quinine resistance. Findings in Malawi were consistent with this interpretation. Artemether and quinine treatment resulted in identical cure rates in children with cerebral malaria.[6] Nonetheless, the length of time to recovery from coma was significantly shorter in the artemether group.

These studies all included relatively small numbers of patients. In this issue of the *Journal,* groups in Vietnam[7] and the Gambia[8] report the results of large studies (560 and 576 patients, respectively) comparing artemether and quinine in the treatment of severe malaria. The study populations and drug regimens differed in the two studies, but the findings were similar. In the Vietnamese study, the patients were at least 15 years of age. Half had cerebral malaria, and the rest had other manifestations of severe malaria (severe anemia, jaundice, renal impairment, hypoglycemia, hyperparasitemia, and shock). They received artemether or quinine every eight hours intramuscularly. In the Gambian study the patients were one to nine years of age (average, four), and all had cerebral malaria. They received intramuscular injections of artemether every 24 hours or quinine every 12 hours.

In the Vietnamese study the case fatality rates were 13 percent in the artemether group and 17 percent in the quinine group ($P=0.16$), with rates of 15 and 16 percent, respectively, among the patients with cerebral malaria. In the Gambian study the case fatality rates were 21 percent in the artemether group and 22 percent in the quinine group ($P=0.8$). In both studies the clearance of parasites was significantly more rapid in the artemether-treated patients, but the length of time to recovery from coma was significantly longer (66 vs. 48 hours in the Vietnamese study [$P=0.003$], and 26 vs. 20 hours in the Gambian study [$P=0.046$]). Convulsions were significantly more common in artemether-treated patients in the Gambia (39 percent vs. 28 percent, $P=0.01$). However, there was no significant difference in the incidence of residual neurologic sequelae between the artemether and quinine groups in the Vietnamese study (1 percent vs. 0.4 percent) or the Gambian study (3 percent vs. 5 percent).

Both studies indicate that artemether is as effective as quinine in reducing the case fatality rate among patients with severe malaria, and neither suggests that artemether is superior to quinine in reducing the

mortality caused by cerebral malaria. Fifty percent of the Vietnamese patients with severe malaria did not have cerebral malaria. The authors did not report the case fatality rate in the subgroup without cerebral malaria. According to my analysis, the reduction in case fatality rates among the artemether-treated patients in this subgroup approached significance (11 percent vs. 18 percent). The authors did perform a multivariate analysis that identified the use of quinine as a risk factor for death, and I assume that the outcome among the patients without cerebral malaria was primarily responsible for this finding. If the slight superiority of artemether over quinine in this subgroup is substantiated, further research will be required to determine whether this is merely a reflection of reduced sensitivity to quinine.

Are there reasons why physicians caring for patients with severe malaria would not use artemisinin derivatives — drugs that can be administered once a day, are well tolerated, and are at least as effective as quinine? This question does not arise in the United States, where this class of drugs is unavailable, although it is likely that arteether, an ethyl ether derivative of artemisinin that is being developed by an international consortium from both the private and public sectors, will be available in a few years. My principal concern is the potential for neurologic toxicity. When administered daily in high doses to dogs and rats, artemether and arteether caused neuropathic lesions in the caudal brain stem.[9] Although in the current reports there was no increase in neurologic sequelae in patients after treatment with artemether, the fact that coma was significantly prolonged in both studies and that there was an increased incidence of convulsions in the artemether group in the Gambian study indicates the need for active investigation of the neurologic side effects of these drugs.

In 1982, the case fatality rate among Thai adults with cerebral malaria treated with intravenous quinine was 17 percent,[5] and in the current study,[7] it was 17 percent among Vietnamese adults with severe malaria who received quinine (16 percent for cerebral malaria). In 14 years, the case fatality rate among patients with severe malaria in Southeast Asia has not decreased. Mortality among Gambian children with cerebral malaria is even higher.[8] The causes of these high rates are poorly understood. The effects of cytokines such as tumor necrosis factor α (TNF-α) and the obstruction of capillaries and venules by adherent parasitized erythrocytes, with resultant local anoxia, are thought to be important.[10] These hypotheses have led to treatment with low-dose[5] and high-dose[11] dexamethasone to inhibit the translation of TNF-α and other inflammatory responses, anti–TNF-α monoclonal antibodies,[8] hyperimmune IgG to reverse microcirculatory obstruction caused by adherent parasitized erythrocytes,[12] and other adjunctive therapy to reduce mortality due to severe malaria. None of these approaches have been effective. Recently, there has been a promising report of the use of pentoxifylline, which inhibits the release of TNF-α from macrophages.[13]

Why is it that none of these logical interventions, when subjected to double-blind, placebo-controlled trials, reduce the case fatality rate? One explanation is that by the time malaria is severe the pathologic changes that cause death are irreversible in some patients. Another is that the pathogenesis of severe disease differs among patients or is so multifactorial in a single patient that a combination of interventions may be required for a clinical effect.

Artemisinin derivatives and quinine are the drugs of choice for treating severe malaria, and artemisinin derivatives are indicated for the treatment of severe malaria caused by parasites with suspected resistance to quinine. Nonetheless, the case fatality rate in severe malaria treated with either of these drugs can be expected to be 10 to 25 percent even under the optimal conditions created by highly trained clinical investigators in formal studies of patients infected with drug-sensitive parasites. Mortality rates will be considerably higher in many health care facilities in the developing world. Neither drug is an adequate treatment for this disease. There is a critical need to identify inexpensive, easily administered antimalarial agents and adjunct therapies that will substantially reduce the case fatality rate in severe malaria.

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The views expressed in this article are those of the author and do not reflect the official policy or position of the Department of the Navy, the Department of Defense, or the U.S. government.

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Letters

In vitro fertilisation for all?

Fertility treatment may be an economic blessing

EDITOR—Ashcroft in his editorial and the National Institute for Clinical Excellence in its consultation document avoid the question of how the value of in vitro fertilisation can be measured both in absolute terms and relative to other health spending priorities.[1 2]

When we read and hear of children born as the result of in vitro fertilisation we should not think, "There's another £15 000 gone." But rather, "There's another couple whose distress has been relieved and who have the opportunity to experience the love and fulfilment of children; there's another human being who has been given the opportunity to live a life; and, there's another person to pay the taxes for our healthcare and pensions when we retire."

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Competing interests: JHW hopes that there will be enough children in the future to pay for his healthcare and pension when he retires.

1 Ashcroft RE. In vitro fertilisation for all? *BMJ* 2003;327:511-2. (6 September.)
2 National Institute for Clinical Excellence. *Fertility: assessment and treatment for people with fertility problems.* London: NICE, 2003. (NICE guideline, second draft for consultation.) (www.nice.org.uk/pdf/Fertility_Fullguideline_2ndconsultation.pdf)

Postcode lottery is more complex than it seems

EDITOR—Inequality of provision of health care because of postcode prescribing is more complex than suggested by Ashcroft.[1] Some differences may indeed result from differences in priorities and values between purchasers, but whether a service is funded depends on rates of ill health and funding locally.

This primary care trust is funded at £10m under its target allocation. We have to prioritise serious medical and psychiatric conditions. This combination of high morbidity and under-resourcing is why we do not fund in vitro fertilisation, not because we place a low value on it.

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Competing interests: None declared.

1 Ashcroft RE. In vitro fertilisation for all? *BMJ* 2003;327:511-2. (6 September.)

Central planning is required

EDITOR—Ashcroft confuses two issues.[1] Local health care provision should reflect the overall profile of the health needs of the local population. However, this fact does not justify a local political process deciding whether childless women with a fertility problem should be given free cycles of in vitro fertilisation treatment.

Suppose that the bulk of the population of a local region is retired. The priorities might then be hip replacements and cataract surgery. Nevertheless, a significant number of couples might have fertility problems that could be treated by in vitro fertilisation. In this case, it would surely be appropriate for those couples to be treated in another region for no more than the costs of travel.

Facilitation of such a process demands exactly the kind of central planning that Ashcroft opposes.

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Society must hear the voices of infertile people

EDITOR—It is interesting that postcode prescribing is due to differences in social values rather than ignorance of the evidence.[1] The National Institute for Clinical Excellence guidelines on infertility endorse the cost effectiveness of in vitro fertilisation, which means that the core problems about providing treatment for infertile couples can now be addressed.[2]

Most people will use NHS resources to control their reproduction at some time. One in six will need help to conceive, and most will have to fund treatment themselves. Society needs to hear the voices of those who have endured infertility. Let local political accountability be based on the equality of their respect and rights.

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Other authors of the original response are Richard Kennedy, Mark Hamilton, Ian Cooke, Neil McClure, Richard Flemming, Umesh Acharya, Elizabeth Lenton, and Gillian Lockwood.

Competing interests: None declared.

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Local decision making has led to inequality

EDITOR—Ashcroft is right to point out that whether fertility treatment is funded by the NHS is essentially a political decision.[1] He is also right to point out that the National Institute for Clinical Excellence is not mandated and does not attempt to settle questions on any basis other than clinical effectiveness. However, his prescription for leaving decisions about funding fertility treatment to a mythically accountable local level is a recipe for perpetuating discrimination and lack of accountability.

The issue of access to fertility treatment is now on the national political agenda partly because of the lack of accountability locally. How are equity and fairness better served by current methods of local accountability than by centrally peer reviewed and clinically effective advice? Either the NHS as a whole subscribes to these basic concepts or we might as well drop the "National."

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1 Ashcroft RE. In vitro fertilisation for all? *BMJ* 2003;327:511-2. (6 September.)

Coeliac disease and subfertility: association is often neglected

EDITOR—Taylor et al provided an excellent series of clinical reviews on subfertility.[1] However, one medical condition that was not mentioned was coeliac disease.

Population screening in the United Kingdom has suggested that coeliac disease has a prevalence of 1%, and this is consistent with other European countries.[2] The relation between coeliac disease and subfertility is well described. Subfertility may be the only presenting feature of coeliac disease.

Two recent studies have shown that the prevalence of unrecognised coeliac disease (as a cause of subfertility) in women presenting to subfertility clinics is in the range of 2.7-3%.[3 4] Not only this, but failure to recognise coeliac disease is also associated with a poorer outcome for the fetus. There may also be male gonadal dysfunction.[5]

Given these findings, surely it is time that clinicians list undiagnosed coeliac disease as an important consideration for preconception advice (under the heading of unrecognised or pre-existing medical problems). The relation between coeliac disease and subfertility continues to be a neglected association in clinical practice.

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Competing interests: None declared.

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High result in prostate specific antigen test

Repeat the test before doing anything further

EDITOR—In their 10-minute consultation Mokete et al discuss how to manage a high result in a prostate specific antigen test.[1]

No man with a prostate specific antigen (PSA) result below 10 should be subjected to the potential morbidity and mortality of prostatic biopsy on a single measurement, irrespective of the presence or otherwise of confounders such as urinary tract infections. General practitioners faced with this result should go back a few issues to Gottlieb's news item.[2 3]

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Competing interests: Like all Western men approaching 60 I have at least one chance in three of having some form of prostate cancer, and like over 50% of British urologists of my age group, I don't know my PSA concentration and don't want to know.

1 Mokete M, Palmer AR, O'Flynn KJ. High result in prostate specific antigen test. *BMJ* 2003;327:379. (16 August.)
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Ten minutes is not enough to deal with the PSA question

EDITOR—The paper by Mokete et al with its electronic responses raises more questions than it answers.[1 2] Perhaps the biggest question is whether a consultation on whether to do a prostate specific antigen (PSA) test is possible in 10 minutes, never mind advising what to do if the result is raised.

The material on the web sites of the Department of Health and national screening committee is extensive. It would take far longer than 10 minutes to read the prostate cancer and PSA fact sheets, let alone go through the questions that men faced with all this information might raise.

The field is hopelessly muddled: there's simply too much conflicting information and advice out there.

Firstly, the benefits of opportunistic PSA testing are yet to be proved.

Secondly, no one should be advised to have a PSA test without a cooling off period to consider all the issues, if necessary on more than one consultation.

Thirdly, when a PSA test result is marginally raised no one should pretend that the way forward is simple or straightforward. There are many choices: from ignoring the test result (difficult to do) to repeating it (the obvious choice, but again, only after the patient has had the opportunity and the time to consider all the implications); from referral to a urologist (who will not have ready answers either) to guided biopsy.

No one knows what the best thing is to do. Perhaps the only conclusion to draw is that the article should have been titled "Not a 10-minute consultation."

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Competing interests: None declared.

1 Mokete M, Palmer AR, O'Flynn KJ. High result in prostate specific antigen test. *BMJ* 2003;327:379. (16 August.)
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Reference intervals for PSA differ

EDITOR—Mokete et al say in their 10-minute consultation that age specific ranges of prostate specific antigen (PSA) have been adopted to improve prostate cancer detection,[1] but the values they quote (unreferenced) are at odds with those promoted by the NHS cancer screening programme cited in the article's Useful reading section. For example, the NHS guidance implies that a PSA >5 ng/ml is suspicious in a man who is 70 or older, but the table in this article says that PSA values up to 6.5 ng/ml are normal in this age group.

Much debate continues around the usefulness, or otherwise, of prostate cancer screening, and PSA action limits differ, depending on which set of guidelines are being followed. However, in the United Kingdom the figures from the cancer screening programme are the ones that NHS primary care clinicians are being asked to follow.

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Repeated testing and free tests might help

EDITOR—The case of a 52 year old man with a total prostate specific antigen (PSA) of 5.7 ng/ml raises at least three other key considerations.[1]

The first is the issue of year to year fluctuation, with a careful longitudinal series showing that over 30% of PSA tests in the 4-10 range returned to normal in following years.[2] The authors suggested repeating the PSA test, perhaps in four to six weeks, before considering biopsy.

The second issue is what constitutes a "high" total PSA value, and whether a PSA threshold for biopsy of above 4.0 is adequate for a man under 60. A recent study found a sensitivity of only 18% using a total PSA >4.0 in the diagnosis of prostate cancer in this age range,[3] and suggested a threshold of 2.5 in younger men.

Finally, interest is increasing in assaying free PSA to inform the need for biopsy, both for total PSA values above and below 4.0 ng/ml.[4 5]

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1 Mokete M, Palmer AR, O'Flynn KJ. High result in prostate specific antigen test. *BMJ* 2003;327:379. (16 August.)
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More on preventing skin cancer

No evolutionary connection exists between skin colour and cancer

EDITOR—Burry reiterates a very important public health message in recommending people cover up and stay out of the sun to avoid skin cancer.[1] I challenge, however, his claims of an evolutionary connection between melanoma and skin colour.

It is true that dark skinned people are less likely to develop melanoma than fair skinned subjects, but melanoma does not affect reproductive fitness because it kills usually in the latter part of life, after those affected have procreated and passed on their genes. It is not, therefore, a notable

factor in human selection and cannot be the cause of the development of the differences in skin colour in Homo sapiens.

Homo sapiens evolved dark skinned in Africa,[2] then started to spread, reaching Europe and Australia 40 000-50 000 years ago.[3] In Australia no selective pressure on skin colour existed and therefore the Australian aboriginal population remained dark.

In Europe, however, selective pressure started to favour individuals with genes for lighter skin. The factor favouring this population change was vitamin D metabolism. Vitamin D can be synthesised only in the presence of ultraviolet light, and dark skin transmits only one tenth of the ultraviolet light that passes through white skin.

Individuals migrating from Africa to Europe started to develop vitamin D deficiency, which affected their reproductive fitness greatly. Individuals who carried genes for reduced skin pigment were favoured, being able to synthesise more vitamin D, leading eventually to a population with a lighter skin colour.[1]

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Competing interests: None declared.

1 Burry JN. Cover up and stay out of sun to prevent skin cancer. *BMJ* 2003;326:1148. (24 May.)
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Author's reply

EDITOR—Procopio alleges that I wrote that people with black skin have been chosen to live in Australia because they are protected against skin cancer. I wrote that black people were chosen by natural selection to live in Australia but did not say why.

Fifteen months after I had begun my systematic inquiry I read for amusement Malthus's essay on population.[1] Being well prepared to appreciate the struggle for existence which everywhere goes on, from the long continued observation of the habits of animals and plants, I was struck at once that under these circumstances favourable variations would tend to be preserved and unfavourable ones to be destroyed.

Aboriginal Australians have succeeded in the struggle for existence for at least 60 000 years because they were able to hunt and gather. Aborigines would not have survived if their skins had not been black in Australia's intense sunlight. Procopio seems to presume that when Australian Aborigines arrived in Australia their skins were black already. It matters little what the colour of their skin was when they arrived, whether their skins were or became black. Australian conditions composed the Darwinian selective forces which ensured that they were fully protected against sunlight by their black skins, which Europeans, who have been chosen by natural selection to live in Europe, are not when they live in Australia.

Europeans in Europe depended on food in their struggle for existence. Vitamin D was essential if they were to survive, and it is reasonable to postulate that that accounts for the colour of their skin. However, again we cannot be certain what the colour of their skin was when they arrived in Europe.

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Competing interests: None declared.

1 Malthus T. An essay on the principle of population. Available at: www.ac.wwu.edu/~stephan/malthus/malthus.0.html (accessed 28 Oct 2003).

Sun avoidance will increase incidence of cancers overall

EDITOR—Fry and Verne in their editorial concluded that sunscreens may create a false sense of security and encourage overexposure to the sun.[1,2] Sunscreens should not be relied on to prevent melanoma. Typical sunscreens only weakly absorb ultraviolet A light, which is 97% of ultraviolet radiation and is nearly as carcinogenic as ultraviolet B light. The typical protection factor for ultraviolet A is 2-4 for chemical sunscreens.[3] Absorption of ultraviolet A is not included in calculation of the sun protection factor.

The authors' advice to avoid the sun would not be the best strategy for reducing overall incidence of cancer. Recommending moderate exposure to the sun would be more prudent.

Solar exposure is the main source of vitamin D. Vitamin D and its metabolites reduce the risk of cancers of the colon,[4,5,w1,w2] breast,[w3–w5] and prostate,[w6–w8] and other cancers.[w9] Since the United Kingdom is located at northern latitudes supplementation of the diet with vitamin D would be helpful, in addition to encouraging moderate exposure to the sun. People in the United Kingdom cannot synthesise vitamin D from November to the end of March, like residents of Boston.[w9] The half life of the storage form of vitamin D is about three weeks, so Britons become deficient by December.

Residents of the United Kingdom should aim for 10-15 minutes a day in the sun when the weather allows, without sunscreen, to allow adequate synthesis of vitamin D. Vitamin D_3 supplementation of children aged 1 year and older and adults at 400 IU per day (10 μg) would be appropriate and would avoid risk of toxicity.[w10] Adults aged 71 and older should receive 600 IU (15 μg) daily.[w11] Sunscreens should be used with caution until products are available that block ultraviolet A with the same degree of protection as ultraviolet B.

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Competing interests: None declared.

References w1-11 are available on bmj.com

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Death in heat waves

Beware of fans ...

EDITOR—In his editorial Keatinge referred to fans as a prevention for heat injury.[1] However, in severe heat, fans can add to the level of heat stress.

Writing about the fatal heat wave of 1995 in Chicago, the US Centers for Disease Control and Prevention noted that fans should not be used for preventing heat related illness in areas with high humidity because increased air movement (such as from fans) is associated with heat stress when the ambient temperature exceeds approximately 100°F (37.8°C) and because fans are not protective at temperatures greater than 90°F (greater than 32.3°C) with humidity greater than 35% (the exact temperature varies with the humidity).[2]

Fans can be extremely effective when combined with the use of a fine water mist in areas of normal or low humidity, but if the temperature and humidity are high, even water mist plus fanning is not reliable for cooling. In such cases people at risk of heat injury are advised to take a cool shower or bath, or seek shelter in an air-conditioned building.

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Competing interests: None declared.

1 Keatinge WR. Death in heat waves. *BMJ* 2003;327:512-3. (6 September.)
2 Centers for Disease Control and Prevention. Heat-related mortality—Chicago, July 1995. *MMWR Morb Mortal Wkly Rep* 1995;44:577-9.

... and injudiciously opening windows

EDITOR—In his editorial on death in heat waves Keatinge surprisingly mentioned that a simple effective measure in protecting elderly people and others against heat is to open a window.[1]

I live in London and at the height of the heat wave the temperature outside in the shade was 37°C. During the few days of the heat wave I maintained the inside of my home at a cool 27°C by ensuring all windows and curtains were closed as soon as the outside temperature was warmer than the inside temperature. In the evening, around 9 pm, the whole procedure was reversed. Once the temperatures outside were similar to those inside the house I would open all curtains and windows to cool down the house and ensure that this coolness was maintained for as long as possible for the following day.

Surely to open the windows when the outside temperature is 37°C and the inside temperature 27°C is absolute madness.

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Competing interests: None declared.

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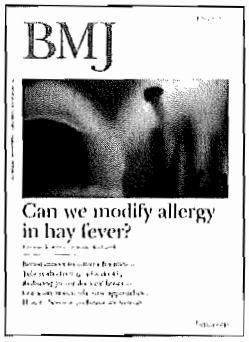
Treatment of seasonal allergic rhinitis



Desensitisation for hay fever works

EDITOR—Desensitisation for hay fever, using conventional high dose extracts, does work[1] and does modify allergy, as reflected by long term remission of disease for at least three years after treatment, and data indicating reduced progression of hay fever to asthma in children.[2,3] On the other hand, the negative results of the study by Radcliffe et al concerning enzyme-potentiated desensitisation using low dose extracts are convincing and seriously question the use of this alternative treatment.[4]

It is unfortunate that your journal cover banner heading did not distinguish the two forms of treatment, with the likely result that general practitioners will be discouraged from referring the small but significant proportion of patients with severe hay fever, unresponsive to nasal corticosteroids and antihistamines, to NHS allergy clinics for consideration of high dose desensitisation.

Equally upsetting was the depiction of a "stargazer," I presume a type of lily, as a cause of hay fever. Hay fever is caused by wind pollinated plants, which include grasses, trees, and weeds. Lilies are insect pollinated. They look nice and smell nice, and they attract insects, but they don't cause hay fever.

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Competing interests: SRD has received research funding, and consultancy and lecture fees from ALK Abello, Horsholm, Denmark, a manufacturer of high-dose vaccines for hay fever.

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Useless versus helpful allergy therapy

EDITOR—Enzyme potentiation has always been controversial and unproved, and Radcliffe et al showed again that it doesn't work.[1] However, the old method of allergy shots that has been around for over 50 years is still quite helpful and safe and endorsed by the World Health Organization as a useful treatment.[2] Allergy desensitisation should be considered in those patients who are frustrated with symptoms that are poorly responsive to medications.

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1 Radcliffe MJ, Lewith GT, Turner RG, Prescott P, Church MK, Holgate ST. Enzyme potentiated desensitisation in treatment of seasonal allergic rhinitis: double blind randomised controlled study. *BMJ* 2003;327:251-4. (2 August.)
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Proactive asthma care does not suit everyone

EDITOR—With reference to the paper by Glasgow et al,[1] prompting general practitioners to provide the 3+ visit plan for children results in children having more consultations, more written asthma plans, but no important differences in outcomes.

Making treatments easier to learn and use may improve health more than prompting doctors and patients to use effective treatments. Conventional structured care that needs children to attend on a regular basis is disease centred and may suit only those children and parents who regard asthma as a chronic disease.[2]

Most children and their parents with mild or moderate asthma manage their asthma as an intermittent acute disorder and may not want a written action plan, or a regular review. A proactive regular review of care can help some patients, but many others only respond to prompts and help when they know they have a problem. They learn most when they have the problem.

A more patient centred approach could be based on health behaviour change,[3] learning,[4] and continuous quality improvement theories.[5] Neglecting these principles delays giving children the structured care they need when they seek help. Clinicians must accept that patients' reactive behaviour is appropriate and structure their care so that whenever, wherever, or whoever the patients consult their immediate needs are met and a reactive regular review of care provided. Proactive care does not suit reactive people.

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Target centred medicine: why it is essential to cheat

EDITOR—D'Sa et al are wrong to believe that only patients are cheated by target centred medicine[1]: we are all cheated, including any gullible members of the public (or politicians) who believe the figures.

For various reasons we have not put our outpatient bookings for our rehabilitation multidisciplinary clinic through the hospital's patient administration system—until we realised that our chronic overload, with outpatient waiting times of up to six months, was not being "seen" and was thus not creating the usual hysteria over "breaches." So we arranged to abandon our personalised booking system and put the appointments through the hospital computer. On discussing this with the relevant managers we discovered that this would make no difference.

Of the 22 patients waiting for an appointment (mean waiting time 23 weeks), only one was from a general practitioner and the rest were tertiary referrals. We were told that only general practitioners' referrals were counted for 17 week wait statistics and tertiary referrals were not. As it happened the general practitioner's referral was to be seen in 16 weeks, so we were not "breaching" at all.

As most tertiary referrals are specifically targeted, often quite serious, and in rehabilitation practice almost the norm it is ludicrous that they can effectively wait indefinitely and no action need be taken, however long they might wait to be seen. It is equally absurd that our service overload is invisible.

So we will cheat. I have written to all our usual tertiary referral sources, asking that they copy the referral letter to the patient's general practitioner, and ask the general practitioner to write a parallel referral.

What a waste of time for the general practitioners. But if it's the only way to show the true picture, then this is what we must do.

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